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SEARCH NOTES

LOGINID:sssptalar1614

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	FEB 25	CA/CAPLUS - Russian Agency for Patents and Trademarks (ROSPATENT) added to list of core patent offices covered
NEWS	4	FEB 28	PATDPAFULL - New display fields provide for legal status data from INPADOC
NEWS	5	FEB 28	BABS - Current-awareness alerts (SDIs) available
NEWS	6	FEB 28	MEDLINE/LMEDLINE reloaded
NEWS	7	MAR 02	GBFULL: New full-text patent database on STN
NEWS	8	MAR 03	REGISTRY/ZREGISTRY - Sequence annotations enhanced
NEWS	9	MAR 03	MEDLINE file segment of TOXCENTER reloaded
NEWS	10	MAR 22	KOREAPAT now updated monthly; patent information enhanced
NEWS	11	MAR 22	Original IDE display format returns to REGISTRY/ZREGISTRY
NEWS	12	MAR 22	PATDPASPC - New patent database available
NEWS	13	MAR 22	REGISTRY/ZREGISTRY enhanced with experimental property tags
NEWS	14	APR 04	EPFULL enhanced with additional patent information and new fields
NEWS	15	APR 04	EMBASE - Database reloaded and enhanced
NEWS	16	APR 18	New CAS Information Use Policies available online

NEWS EXPRESS	JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

* * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 10:45:15 ON 22 APR 2005

=> file hcaplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'HCAPLUS' ENTERED AT 10:45:27 ON 22 APR 2005
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

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FILE COVERS 1907 - 22 Apr 2005 VOL 142 ISS 18
FILE LAST UPDATED: 21 Apr 2005 (20050421/ED)

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This file contains CAS Registry Numbers for easy and accurate substance identification.

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=> e heirler h/au
E1            2     HEIRLER F/AU
E2            1     HEIRLER FLORIAN/AU
E3            0 --> HEIRLER H/AU
E4            2     HEIRLER HORST/AU
E5            2     HEIRLINGS L/AU
E6            1     HEIRMAN A/AU
E7            1     HEIRMAN ANS/AU
E8            1     HEIRMAN C/AU
E9           28     HEIRMAN CARLO/AU
E10           1     HEIRMAN FREDERIC/AU
E11           2     HEIRMAN G/AU
E12           3     HEIRMAN INGEBORG/AU

=> s e4
L1            2 "HEIRLER HORST"/AU

=> d l1 1-2 ibib ed abs

L1   ANSWER 1 OF 2  HCAPLUS  COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER:         2004:427581  HCAPLUS
DOCUMENT NUMBER:          140:422820
TITLE:                    Use of medium chain triglycerides for the nutritional
                          optimization of the lipid composition of a dietetic
                          product for diabetics
INVENTOR(S):              Heirler, Horst
PATENT ASSIGNEE(S):       Horst Heirler Projekte Ernaehrungmedizinoekologie,
                          Germany
SOURCE:                   Eur. Pat. Appl., 15 pp.
                          CODEN: EPXXDW
DOCUMENT TYPE:            Patent
LANGUAGE:                 German
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.        DATE
     ---------------      ----   --------    --------------------   --------
     EP 1421858            A1    20040526    EP 2003-26659          20031119
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
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DE 10254584	A1	20040609	DE 2002-10254584		20021122
US 2004151757	A1	20040805	US 2003-717990		20031121
PRIORITY APPLN. INFO.:			DE 2002-10254584	A	20021122

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:899191 HCAPLUS
DOCUMENT NUMBER: 123:296653
TITLE: Dietetic fatty acid mixture for treatment of malabsorption syndrome.
INVENTOR(S): **Heirler, Horst**
PATENT ASSIGNEE(S): Germany
SOURCE: Ger., 4 pp.
CODEN: GWXXAW
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 4417851	C1	19951005	DE 1994-4417851	19940520
EP 682879	A1	19951122	EP 1995-107610	19950518
EP 682879	B1	20001108		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
AT 197380	E	20001111	AT 1995-107610	19950518
PT 682879	T	20010228	PT 1995-107610	19950518
ES 2153443	T3	20010301	ES 1995-107610	19950518
FI 9502465	A	19951121	FI 1995-2465	19950519
NO 9501992	A	19951121	NO 1995-1992	19950519
NO 312393	B1	20020506		
GR 3035094	T3	20010330	GR 2000-402782	20001218
PRIORITY APPLN. INFO.:			DE 1994-4417851 A	19940520

ED Entered STN: 07 Nov 1995
AB The title mixture comprises: medium-chain fatty acids 70-90, α-linoleic acid 3.8-13.4, and α-linolenic acid 3-8%. Optional components are γ-linolenic acid, fat-soluble vitamins and mineral elements,.

=> file medline biosis hcaplus embase wpids

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	7.75	7.96
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-1.46	-1.46

FILE 'MEDLINE' ENTERED AT 10:46:14 ON 22 APR 2005

FILE 'BIOSIS' ENTERED AT 10:46:14 ON 22 APR 2005

FILE 'HCAPLUS' ENTERED AT 10:46:14 ON 22 APR 2005

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FILE 'EMBASE' ENTERED AT 10:46:14 ON 22 APR 2005
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diabetes" or "type II diabetes" or (diabetes(W)insipidus) or (glucose(W)intoleran?)
or hyperglycem? or (impaired glucose toleran?)
   4 FILES SEARCHED...
L2      624438 (DIABETES(W) MELLITUS) OR "TYPE 1 DIABETES" OR "TYPE 2 DIABETES"
               OR "TYPE I DIABETES" OR "TYPE II DIABETES" OR (DIABETES(W)
               INSIPIDUS) OR (GLUCOSE(W) INTOLERAN?) OR HYPERGLYCEM? OR (IMPAIR
               ED GLUCOSE TOLERAN?)

=> s (medium(W)chain(W)triglyceride?) or triacylglycerol? or triglyceride? or "MCT"
or "MCTs" or (capric acid?) or (caprylic acid?) or (decanoic acid?) or octanoate?
or (octanoic acid?)
   3 FILES SEARCHED...
L3      316701 (MEDIUM(W) CHAIN(W) TRIGLYCERIDE?) OR TRIACYLGLYCEROL? OR TRIGLY
               CERIDE? OR "MCT" OR "MCTS" OR (CAPRIC ACID?) OR (CAPRYLIC ACID?)
               OR (DECANOIC ACID?) OR OCTANOATE? OR (OCTANOIC ACID?)

=> s (oleic acid?) or "9-octadecenoic acid" or oleate? or (olive oil?) or (rape?
oil?) or (canola oil?) or (monounsaturated(W)fatty(W)acid?) or (monoene?) or "
MISMATCHED QUOTE 'OR "'
Quotation marks (or apostrophes) must be used in pairs,
one before and one after the expression you are setting
off or masking.

=> s (oleic acid?) or "9-octadecenoic acid" or oleate? or (olive oil?) or (rape?
oil?) or (canola oil?) or (monounsaturated(W)fatty(W)acid?) or (monoene?) or "MUFA"
or "MUFAs"
   4 FILES SEARCHED...
L4      198146 (OLEIC ACID?) OR "9-OCTADECENOIC ACID" OR OLEATE? OR (OLIVE
               OIL?) OR (RAPE? OIL?) OR (CANOLA OIL?) OR (MONOUNSATURATED(W)
               FATTY(W) ACID?) OR (MONOENE?) OR "MUFA" OR "MUFAS"

=> s (linoleic acid?) or linoleate? or "9,12-octadecadienoic acid" or (linoelaidic
acid?) or (sunflower oil?) or (rape? oil?) or (omega(W)"6"(W)fatty(W)acid?)
   3 FILES SEARCHED...
L5      122681 (LINOLEIC ACID?) OR LINOLEATE? OR "9,12-OCTADECADIENOIC ACID"
               OR (LINOELAIDIC ACID?) OR (SUNFLOWER OIL?) OR (RAPE? OIL?) OR
               (OMEGA(W)"6"(W) FATTY(W) ACID?)

=> s (ω)(W)"6"(W)fatty(W)acid? or (double(W)unsaturated(W)triglyceride?)
L6        2949 (Ω)(W)"6"(W) FATTY(W) ACID? OR (DOUBLE(W) UNSATURATED(W)
               TRIGLYCERIDE?)

=> s ((α)(W)linolenic(W)acid?) or (linolenic acid?) or linolenate? or
"9,12,15-octadecatrienoic acid" or (rape? oil?) or (linseed oil?) or
(omega(W)"3"(W)fatty(W)acid?) or ((ω)(W)"3"(W)fatty(W)acid?)
   2 FILES SEARCHED...
L7       85651 ((A)(W) LINOLENIC(W) ACID?) OR (LINOLENIC ACID?) OR LINOLE
               NATE? OR "9,12,15-OCTADECATRIENOIC ACID" OR (RAPE? OIL?) OR
               (LINSEED OIL?) OR (OMEGA(W)"3"(W) FATTY(W) ACID?) OR ((Ω)(
               W)"3"(W) FATTY(W) ACID?)

=> s (triple(W)unsaturated(W)triglyceride?)
L8           1 (TRIPLE(W) UNSATURATED(W) TRIGLYCERIDE?)

=> s eicosapentaen? or eicosapentaenoic acid? or timnodonic acid? or "EPA"
L9       52757 EICOSAPENTAEN? OR EICOSAPENTAENOIC ACID? OR TIMNODONIC ACID? OR
               "EPA"
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=> s docosahexaen? or docosahexaenoic acid? or "DHA" or (fish oil?) or ((shellfish
or tuna or mackerel or salmon or menhaden or menhadin or anchovy or herring or

=> s (saturated(W)long(W)chain(W)(triglyceride? or triacylglycerol?)) or
(long(W)chain(W) (triglyceride? or triacylglycerol?)) or "LCT" or "LCTs"
L11       4405 (SATURATED(W) LONG(W) CHAIN(W)(TRIGLYCERIDE? OR TRIACYLGLYCEROL?
               )) OR (LONG(W) CHAIN(W) (TRIGLYCERIDE? OR TRIACYLGLYCEROL?)) OR
               "LCT" OR "LCTS"

=> s emulsifier or emulsif?
L12     152425 EMULSIFIER OR EMULSIF?

=> s monoglyceride? or monoacylglycerol? or diglyceride? or diacylglycerol?
L13      69467 MONOGLYCERIDE? OR MONOACYLGLYCEROL? OR DIGLYCERIDE? OR DIACYLGLY
               CEROL?

=> s "vitamin A" or retinol? or retinyl palmitate? or "vitamin D" or
cholecalciferol? or "vitamin E" or tocopherol? or tocotrienol? or tocopherol
acetate? or "vitamin C" or ascorbyl palmitate?
L14     421736 "VITAMIN A" OR RETINOL? OR RETINYL PALMITATE? OR "VITAMIN D" OR
               CHOLECALCIFEROL? OR "VITAMIN E" OR TOCOPHEROL? OR TOCOTRIENOL?
               OR TOCOPHEROL ACETATE? OR "VITAMIN C" OR ASCORBYL PALMITATE?

=> s carotin? or (beta(W)(carotin? or carotene?)) or ((β)(W) (carotin? or
carotene?)) or bellacarotin? or carotaben or provatene? or solatene? or vetoron?
L15      48454 CAROTIN? OR (BETA(W)(CAROTIN? OR CAROTENE?)) OR ((B)(W)
               (CAROTIN? OR CAROTENE?)) OR BELLACAROTIN? OR CAROTABEN OR PROVAT
               ENE? OR SOLATENE? OR VETORON?

=> d cost
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CONNECT CHARGES                                         89.15      91.69
NETWORK CHARGES                                          2.10       2.22
DISPLAY CHARGES                                          0.00       5.30
                                                    -------    -------
FULL ESTIMATED COST                                     91.25      99.21

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                      0.00      -1.46

IN FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS'
AT 11:07:33 ON 22 APR 2005


=> s flavoring? or (butter(W)flavor?) or rosemary? or (rosemary(W)extract?) or
rosmarinus? or (rosmarinus(W)extract?)
L16      27388 FLAVORING? OR (BUTTER(W) FLAVOR?) OR ROSEMARY? OR (ROSEMARY(W)
               EXTRACT?) OR ROSMARINUS? OR (ROSMARINUS(W) EXTRACT?)

=> s retinyl palmitate? or "vitamin D3" or cholecalciferol? or "vitamin E" or
"RRR-α-tocopheryl acetate" or ascorbyl palmitate?
L17     130972 RETINYL PALMITATE? OR "VITAMIN D3" OR CHOLECALCIFEROL? OR "VITAM
               IN E" OR "RRR-A-TOCOPHERYL ACETATE" OR ASCORBYL PALMITATE?

=> s "vitamin B6" or picoline? or pyridoxine? or pyridoxal? or pyridoxamine? or
"vitamin B12" or cobalamin? or cyanocobalamin? or folic acid? or (pteroylglutamic
acid?) or folacin? or folvite? or homofolic acid?
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   4 FILES SEARCHED...
L18     197160 "VITAMIN B6" OR PICOLINE? OR PYRIDOXINE? OR PYRIDOXAL? OR PYRIDO
               XAMINE? OR "VITAMIN B12" OR COBALAMIN? OR CYANOCOBALAMIN? OR



thiamine? or aneurin? or "vitamin B2" or riboflavin? or flavin? or "vitamin G" or
niacin? or nicotine amide? or nicotinic acid? or "3-pyridinecarboxylic acid" or
enduracin? or induracin? or nicamin? or nicotinate?
   4 FILES SEARCHED...
L19     404698 "VITAMIN C" OR ASCORBIC ACID? OR ASCORBYL PALMITATE? OR "VITAMIN
                B1" OR THIAMINE? OR ANEURIN? OR "VITAMIN B2" OR RIBOFLAVIN? OR
               FLAVIN? OR "VITAMIN G" OR NIACIN? OR NICOTINE AMIDE? OR NICOTINI
               C ACID? OR "3-PYRIDINECARBOXYLIC ACID" OR ENDURACIN? OR INDURACI
               N? OR NICAMIN? OR NICOTINATE?

=> s zinc? or chrom? or manganese?
L20    4846639 ZINC? OR CHROM? OR MANGANESE?

=> d cost
COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                 ENTRY    SESSION
CONNECT CHARGES                                 119.92     122.46
NETWORK CHARGES                                   2.88       3.00
DISPLAY CHARGES                                   0.00       5.30
                                                -------    -------
FULL ESTIMATED COST                             122.80     130.76

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)    SINCE FILE      TOTAL
                                                 ENTRY    SESSION
CA SUBSCRIBER PRICE                               0.00      -1.46

IN FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS'
AT 11:15:00 ON 22 APR 2005


=> d his

     (FILE 'HOME' ENTERED AT 10:45:15 ON 22 APR 2005)

     FILE 'HCAPLUS' ENTERED AT 10:45:27 ON 22 APR 2005
               E HEIRLER H/AU
L1             2 S E4

     FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS' ENTERED AT 10:46:14 ON 22
     APR 2005
L2        624438 S (DIABETES(W)MELLITUS) OR "TYPE 1 DIABETES" OR "TYPE 2 DIABETE
L3        316701 S (MEDIUM(W)CHAIN(W)TRIGLYCERIDE?) OR TRIACYLGLYCEROL? OR TRIGL
L4        198146 S (OLEIC ACID?) OR "9-OCTADECENOIC ACID" OR OLEATE? OR (OLIVE O
L5        122681 S (LINOLEIC ACID?) OR LINOLEATE? OR "9,12-OCTADECADIENOIC ACID"
L6          2949 S (Ω)(W)"6"(W)FATTY(W)ACID? OR (DOUBLE(W)UNSATURATED(W)TR
L7         85651 S ((A)(W)LINOLENIC(W)ACID?) OR (LINOLENIC ACID?) OR LINOL
L8             1 S (TRIPLE(W)UNSATURATED(W)TRIGLYCERIDE?)
L9         52757 S EICOSAPENTAEN? OR EICOSAPENTAENOIC ACID? OR TIMNODONIC ACID?
L10        60428 S DOCOSAHEXAEN? OR DOCOSAHEXAENOIC ACID? OR "DHA" OR (FISH OIL?
L11         4405 S (SATURATED(W)LONG(W)CHAIN(W)(TRIGLYCERIDE? OR TRIACYLGLYCEROL
L12       152425 S EMULSIFIER OR EMULSIF?
L13        69467 S MONOGLYCERIDE? OR MONOACYLGLYCEROL? OR DIGLYCERIDE? OR DIACYL
L14       421736 S "VITAMIN A" OR RETINOL? OR RETINYL PALMITATE? OR "VITAMIN D"
L15        48454 S CAROTIN? OR (BETA(W)(CAROTIN? OR CAROTENE?)) OR ((B)(W)
L16        27388 S FLAVORING? OR (BUTTER(W)FLAVOR?) OR ROSEMARY? OR (ROSEMARY(W)
L17       130972 S RETINYL PALMITATE? OR "VITAMIN D3" OR CHOLECALCIFEROL? OR "VI
L18       197160 S "VITAMIN B6" OR PICOLINE? OR PYRIDOXINE? OR PYRIDOXAL? OR PYR
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L19      404698 S "VITAMIN C" OR ASCORBIC ACID? OR ASCORBYL PALMITATE? OR "VITA
L20     4846639 S ZINC? OR CHROM? OR MANGANESE?



L22        699 L21 AND L4

=> s l22 and (l5 or l6 or l7 or l8)
L23        204 L22 AND (L5 OR L6 OR L7 OR L8)

=> s l23 and (l9 or l10)
L24         73 L23 AND (L9 OR L10)

=> s l24 and l11
L25          1 L24 AND L11

=> d l25

L25  ANSWER 1 OF 1  WPIDS  COPYRIGHT 2005 THE THOMSON CORP on STN
AN   2004-422255 [40]   WPIDS
DNC  C2004-158703
TI   Dietetic therapy of diabetes mellitus in adults or
     adolescents, using medium-chain triglycerides
     , e.g. incorporated in margarine or edible oil, to regulate and optimize
     metabolism.
DC   B05 D13
IN   HEIRLER, H
PA   (HEIR-N) HEIRLER PROJEKTE ERNAEHRUNG MEDIZIN OEKO
CYC  32
PI   EP 1421858      A1 20040526 (200440)* GE   15    A23L001-30
         R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV
            MC MK NL PT RO SE SI SK TR
     DE 10254584     A1 20040609 (200440)             A23L001-30
     US 2004151757   A1 20040805 (200452)             A61K031-225
ADT  EP 1421858 A1 EP 2003-26659 20031119; DE 10254584 A1 DE 2002-10254584
     20021122; US 2004151757 A1 US 2003-717990 20031121
PRAI DE 2002-10254584     20021122
IC   ICM  A23L001-30; A61K031-225
     ICS  A23D007-00; A23L001-03; A23L001-29; A23L001-302; A23L001-304;
          A61K031-20; A61K047-00


=> s l24 and (l12 or l13 or l14 or l15 or l16)
L26         14 L24 AND (L12 OR L13 OR L14 OR L15 OR L16)

=> dup rem l26
PROCESSING COMPLETED FOR L26
L27          11 DUP REM L26 (3 DUPLICATES REMOVED)
                ANSWER '1' FROM FILE MEDLINE
                ANSWERS '2-5' FROM FILE HCAPLUS
                ANSWERS '6-9' FROM FILE EMBASE
                ANSWERS '10-11' FROM FILE WPIDS

=> d l27 1-11 ibib ed abs

L27  ANSWER 1 OF 11     MEDLINE on STN                  DUPLICATE 3
ACCESSION NUMBER:   94168761     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 8123291
TITLE:              High dosage vitamin E effect on
                    oxidative status and serum lipids distribution in
                    streptozotocin-induced diabetic rats.
AUTHOR:             Douillet C; Chancerelle Y; Cruz C; Maroncles C; Kergonou J
```

F; Renaud S; Ciavatti M
CORPORATE SOURCE: National Institute of Health and Medical Research, Unit 63, Lyon-Bron, France

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199404
ENTRY DATE: Entered STN: 19940420
Last Updated on STN: 19940420
Entered Medline: 19940411

ED Entered STN: 19940420
Last Updated on STN: 19940420
Entered Medline: 19940411

AB This study was performed to determine whether **vitamin E** supplementation in streptozotocin-induced diabetic rats treated by insulin could reduce serum oxidation markers (malondialdehyde: MDA, Schiff bases, anti-protein-MDA adduct antibodies) and modulate lipid changes. After 10 weeks, diabetes induced in rats a significant increase in Schiff bases ($P < 0.006$) and anti-protein-MDA adduct antibodies ($P < 0.01$). These alterations were accompanied by a significant rise in serum free fatty acids (225%), **triglycerides** (35%), and phospholipids (30%) and changes in fatty acid distribution in these fractions and in cholesterol esters. **Vitamin E** supplementation in diabetic rats reduced Schiff bases and anti-protein-MDA adduct antibodies and tended to restore the fatty acid profile close to control rats without decreasing quantitatively serum lipids enhanced by diabetes. Concerning fatty acids, **vitamin E** chiefly reduced stearic acid (C18:0) in free fatty acids, cholesterol esters, and phospholipids and cancelled the decrease in low molecular **triglycerides** observed in diabetic rats. Furthermore, **vitamin E** maintained the ratio of monounsaturated and polyunsaturated fatty acids, particularly with respect to **oleic acid** (C18:1), dihomo-gamma-**linolenic acid** (C20:3 n-6), **eicosapentaenoic** (C20:5 n-3), and docosapentaenoic acid (C22:5 n-3), in serum phospholipids. These changes observed in **vitamin E** supplemented rats, compared to **vitamin E**-untreated diabetic rats, could favor prevention of accelerated atherogenesis. Particularly, the decrease of serum peroxides and enhancement in phospholipid fatty acids (C20:3 n-6, C20:5 n-3, and C22:5 n-3) could induce the preferential formation of prostaglandins (PGE1, PGI2, PGI3) which are protective in cardiovascular diseases.

L27 ANSWER 2 OF 11 HCAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2004:681501 HCAPLUS
DOCUMENT NUMBER: 141:185110
TITLE: Oils enriched with **diacylglycerols** and phytosterol esters for use in the reduction of cholesterol and **triglycerides**
INVENTOR(S): Platt, Dorit; Pelled, Dori; Shulman, Avidor
PATENT ASSIGNEE(S): Enzymotec Ltd., Israel
SOURCE: PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004069150	A2	20040819	WO 2004-IL131	20040210

WO 2004069150 A3 20040923
W: AE, AE, AG, AL, AL, AM, AM, AM, AT, AT, AU, AZ, AZ, BA, BB, BG,
BG, BR, BR, BW, BY, BY, BZ, BZ, CA, CH, CN, CN, CO, CO, CR, CR
MZ, MZ, NA, NI
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE,
BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU,
MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN,
GQ, GW, ML, MR, NE, SN, TD, TG, BF, BJ, CF, CG, CI, CM, GA, GN,
GQ, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: IL 2003-154381 A 20030210
IL 2003-155136 A 20030327
ED Entered STN: 20 Aug 2004
AB The present invention relates to the use of a composition comprising a combination of **diacylglycerol**(s) (DAG), mainly 1,3-**diacylglycerol**(s), and phytosterol and/or phytostanol ester(s) (PSE) dissolved or dispersed in edible oil and/or edible fat, particularly olive, canola and **fish oil**, in the manufacture of nutritional supplements and orally administrable pharmaceutical prepns. which are capable of reducing blood levels of both cholesterol and **triglycerides** and/or for lowering serum, serum LDL and macrophage oxidation levels, inhibiting the formation of foam cells and/or preventing the deleterious effects generated by lipid-induced oxidative stress. In addition, the composition of the invention, as well as the pharmaceutical prepns.
thereof, is suitable for the treatment and prevention of conditions related to atherosclerosis, such as cardiovascular disease (CVD), coronary heart disease (CHD) and **diabetes mellitus**.
Olive oil containing 1,3-**diacylglycerols**, phytosterols and phytosterol esters (olive MultOil) gave improved antioxidative effect against macrophage lipid peroxidn. in apolipoprotein E deficient mice compared with **olive oil** or **olive oil** plus phytosterols.

L27 ANSWER 3 OF 11 HCAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 2004:427581 HCAPLUS
DOCUMENT NUMBER: 140:422820
TITLE: Use of **medium chain triglycerides** for the nutritional optimization of the lipid composition of a dietetic product for diabetics
INVENTOR(S): Heirler, Horst
PATENT ASSIGNEE(S): Horst Heirler Projekte Ernaehrungmedizinoekologie, Germany
SOURCE: Eur. Pat. Appl., 15 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1421858	A1	20040526	EP 2003-26659	20031119
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
DE 10254584	A1	20040609	DE 2002-10254584	20021122
US 2004151757	A1	20040805	US 2003-717990	20031121

PRIORITY APPLN. INFO.: DE 2002-10254584 A 20021122
ED Entered STN: 27 May 2004
AB **Medium-chain triglycerides** are used at

10-30% of total fat with at least one simple unsatd. fatty acid such as **oleic acid** in manufacture of lipid-based foods, especially dietetic foods used for treatment of diabetes mellitus

ACCESSION NUMBER: 2003:656425 HCAPLUS
DOCUMENT NUMBER: 139:159947
TITLE: Method for activating the lipid catabolic metabolism in enteric epithelium and improving the lipid metabolism in enteric epithelium
INVENTOR(S): Hase, Tadashi; Murase, Takatoshi; Watanabe, Hiroyuki; Kondo, Hidehiko
PATENT ASSIGNEE(S): Kao Corporation, Japan
SOURCE: U.S. Pat. Appl. Publ., 21 pp., Cont.-in-part of U.S. Ser. No. 131,188.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003158257	A1	20030821	US 2002-238720	20020911
JP 2002322052	A2	20021108	JP 2001-129847	20010426
US 2003096866	A1	20030522	US 2002-131188	20020425
PRIORITY APPLN. INFO.:			JP 2001-129847 A	20010426
			US 2002-131188 A2	20020425

ED Entered STN: 22 Aug 2003
AB Disclosed are a method for activating lipid metabolism in the small intestine epithelium and also a method for promoting accumulation of fatty acids into the small intestine epithelium, each of which features administering an effective amount of a **diacylglycerol**. Also disclosed are methods for improving various symptoms in diabetes, which have ingesting a **diacylglycerol**. Ingestion of the **diacylglycerol** leads to accumulation of the fatty acids in the small intestine. The fatty acids so accumulated promote induction of β-oxidation, thereby not only activating lipid catabolism but also making it difficult to allow lipids to accumulate as **triacylglycerols**. This series of actions eventually results in development of lowering action for blood remnant-like lipoprotein level and also lowering action for blood leptin level, and hence, lipid metabolism is improved. Further, energy consumption is enhanced by promoting the induction of β-oxidation and activating lipid catabolism.

L27 ANSWER 5 OF 11 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1991:5246 HCAPLUS
DOCUMENT NUMBER: 114:5246
TITLE: Metabolic and hemostatic consequences of dietary fiber and n-3 fatty acids in black type 2 (NIDDM) diabetic subjects: a placebo controlled study
AUTHOR(S): Silvis, Nelly; Vorster, Hester H.; Mollentze, Willie F.; De Jager, Johan; Huisman, Hugo W.
CORPORATE SOURCE: Dep. Diet., Potchefstroom Univ. Christ. Higher Educ., Potchefstroom, 2520, RSA, S. Afr.
SOURCE: International Clinical Nutrition Review (1990), 10(3), 362-80
CODEN: ICNRDJ; ISSN: 0813-9008
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 12 Jan 1991
AB Metabolic and hemostatic effects of a fiber concentrate (11.4 g fiber/day) and

an **eicosapentaenoic acid (EPA)** supplement (1.4 g/day) was examined in 63 black Type 2 diabetics in a cross-over, placebo (olive oil) controlled study. Dietary

fiber concentrate was reasonably good, and as checked by capsule count, it was excellent with **EPA** supplementation. Although there was no improvement in glycemic control, the expected beneficial effects of dietary fiber on total serum cholesterol and especially serum high-d. lipoprotein cholesterol, as well as an increase in in vitro clotting times, were observed There were no adverse effects on serum mineral status. Results indicated possible hemolysis of red blood cells in the first group that received **EPA** supplementation (decreased levels of Hb, serum, and increased levels of serum bilirubin, serum unconjugated bilirubin, and serum lactate dehydrogenase). After an addnl. 100 IU of **vitamin E** per day was added to the **EPA** supplementation during phase II, a tendency towards the same changes was still noticed. In this group, serum **triglycerides** decreased significantly (36%) and bleeding times were prolonged (31%). However, this dosage of **EPA** supplementation was associated with potentially detrimental changes in glycemic control, fibrinogen concns., and factor VII coagulant activity. It is concluded that the use of a fiber supplement in black diabetic patients should be discouraged.

L27 ANSWER 6 OF 11 EMBASE on STN

ACCESSION NUMBER: 2002425432 EMBASE
TITLE: Fish consumption, **fish oil**, **omega-3 fatty acids**, and cardiovascular disease.
AUTHOR: Kris-Etherton P.M.; Harris W.S.; Appel L.J.
SOURCE: Circulation, (19 Nov 2002) Vol. 106, No. 21, pp. 2747-2757. Refs: 119
ISSN: 0009-7322 CODEN: CIRCAZ
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 017 Public Health, Social Medicine and Epidemiology
018 Cardiovascular Diseases and Cardiovascular Surgery
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20021205
Last Updated on STN: 20021205
ED Entered STN: 20021205
Last Updated on STN: 20021205

AB **Omega-3 fatty acids** have been shown in epidemiological and clinical trials to reduce the incidence of CVD. Large-scale epidemiological studies suggest that individuals at risk for CHD benefit from the consumption of plant- and marine-derived **omega-3 fatty acids**, although the ideal intakes presently are unclear. Evidence from prospective secondary prevention studies suggests that **EPA+DHA** supplementation ranging from 0.5 to 1.8 g/d (either as fatty fish or supplements) significantly reduces subsequent cardiac and all-cause mortality. For .**alpha.-linolenic acid**, total intakes of ≈1.5 to 3 g/d seem to be beneficial. Collectively, these data are supportive of the recommendation made by the AHA Dietary Guidelines to include at least two servings of fish per week (particularly fatty fish). In addition, the data support inclusion of vegetable oils (eg, soybean, canola, walnut, flaxseed) and food sources (eg, walnuts, flaxseeds) high in .**alpha.-linolenic**

acid in a healthy diet for the general population (Table 5). The fish recommendation must be balanced with concerns about environmental pollutants, in particular PCB and methylmercury, described in state and

supplements can reduce cardiac events (eg, death, nonfatal MI, nonfatal stroke) and decrease progression of atherosclerosis in coronary patients. However, additional studies are needed to confirm and further define the health benefits of **omega-3 fatty acid** supplements for both primary and secondary prevention. For example, placebo-controlled, double-blind RCTs are needed to document both the safety and efficacy of **omega-3 fatty acid** supplements in both high-risk patients (eg, patients with **type 2 diabetes**, dyslipidemia, and hypertension, and smokers) and coronary patients on drug therapy. Mechanistic studies on their apparent effects on sudden death are also needed. A dietary (ie, food-based) approach to increasing **omega -3 fatty acid** intake is preferable. Still, for patients with coronary artery disease, the dose of omega-3 (≈1 g/d) may be greater than what can readily be achieved through diet alone (Table 5). These individuals, in consultation with their physician, could consider supplements for CHD risk reduction. Supplements also could be a component of the medical management of hypertriglyceridemia, a setting in which even larger doses (2 to 4 g/d) are required (Table 5). The availability of high-quality **omega-3 fatty acid** supplements, free of contaminants, is an important prerequisite to their extensive use.

L27 ANSWER 7 OF 11 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2002403663 EMBASE
TITLE: Metabolic syndrome X is common in South Asians, but why and how?.
AUTHOR: Das U.N.
CORPORATE SOURCE: Dr. U.N. Das, EFA Sciences LLC, 1420 Providence Highway, Norwood, MA 02062, United States. undurti@hotmail.com
SOURCE: Nutrition, (2002) Vol. 18, No. 9, pp. 774-776.
Refs: 31
ISSN: 0899-9007 CODEN: NUTRER
PUBLISHER IDENT.: S 0899-9007(02)00826-2
COUNTRY: United States
DOCUMENT TYPE: Journal; Note
FILE SEGMENT: 022 Human Genetics
029 Clinical Biochemistry
LANGUAGE: English
ENTRY DATE: Entered STN: 20021202
Last Updated on STN: 20021202
ED Entered STN: 20021202
Last Updated on STN: 20021202
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L27 ANSWER 8 OF 11 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2002193935 EMBASE
TITLE: **Eicosapentaenoic acid** and **docosahexaenoic acid** from **fish oils**: Differential associations with lipid responses.
AUTHOR: Leigh-Firbank E.C.; Minihane A.M.; Leake D.S.; Wright J.W.; Murphy M.C.; Griffin B.A.; Williams C.M.
CORPORATE SOURCE: Dr. A.M. Minihane, Hugh Sinclair Unit of Human Nutr., University of Reading, Reading, United Kingdom.

minihane@reading.ac.uk
SOURCE: British Journal of Nutrition, (2002) Vol. 87, No. 5, pp. 435-445

FILE SEGMENT: 029 Clinical Biochemistry
018 Cardiovascular Diseases and Cardiovascular Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20020613
Last Updated on STN: 20020613
ED Entered STN: 20020613
Last Updated on STN: 20020613

AB **Fish-oil** supplementation can reduce circulating **triacylglycerol** (TG) levels and cardiovascular risk. This study aimed to assess independent associations between changes in platelet **eicosapentaenoic acid** (**EPA**) and **docosahexaenoic acid** (**DHA**) and fasting and postprandial (PP) lipoprotein concentrations and LDL oxidation status, following **fish-oil** intervention. Fifty-five mildly hypertriacylglycerolaemic (TG 1.5-4.0 mmol/l) men completed a double-blind placebo controlled cross over study, where individuals consumed 6 g **fish oil** (3 g **EPA** + **DHA**) or 6 g **olive oil** (placebo)/d for two 6-week intervention periods, with a 12-week wash-out period in between. **Fish-oil** intervention resulted in a significant increase in the platelet phospholipid **EPA** (+491 %, P<0.001) and **DHA** (+44 %, P<0.001) content and a significant decrease in the arachidonic acid (-10 %, P<0.001) and γ- **linolenic acid** (-24%, P<0.001) levels. A 30 % increase in ex vivo LDL oxidation (P<0.001) was observed. In addition, **fish oil** resulted in a significant decrease in fasting and PP TG levels (P<0.001), PP non-esterified fatty acid (NEFA) levels, and in the percentage LDL as LDL-3 (P=0.040), and an increase in LDL-cholesterol (P=0.027). In multivariate analysis, changes in platelet phospholipid **DHA** emerged as being independently associated with the rise in LDL-cholesterol, accounting for 16 % of the variability in this outcome measure (P=0.030). In contrast, increases in platelet **EPA** were independently associated with the reductions in fasting (P=0.046) and PP TG (P=0.023), and PP NEFA (P=0.015), explaining 15-20 % and 25 % of the variability in response respectively. Increases in platelet **EPA** + **DHA** were independently and positively associated with the increase in LDL oxidation (P=0.011). **EPA** and **DHA** may have differential effects on plasma lipids in mildly hypertriacylglycerolaemic men.

L27 ANSWER 9 OF 11 EMBASE on STN
ACCESSION NUMBER: 94332026 EMBASE
DOCUMENT NUMBER: 1994332026
TITLE: Dyslipidemia and coronary artery disease.
AUTHOR: Kuo P.T.
CORPORATE SOURCE: VA Medical Center, 2002 Holocombe Blvd.,Houston, TX 77030, United States
SOURCE: Clinical Cardiology, (1994) Vol. 17, No. 10, pp. 519-527.
ISSN: 0160-9289 CODEN: CLCADC
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 006 Internal Medicine
017 Public Health, Social Medicine and Epidemiology
018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index

038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Genetically determined and metabolically induced disturbances in lipid metabolism, as manifested in several types of dyslipidemia, have been shown to be causally related to the development of coronary artery disease (CAD). A diversity of clinical and angiographic studies has been made to evaluate the linkage between plasma lipid-control therapy in the development of initial and recurrent cardiovascular events. The plan of treatment invariably begins with a low-fat, low-cholesterol diet before initiation of drug therapy. However, many patients have difficulty in adhering to the low-fat diet. Fortunately, metabolic studies show that foods which contain fats rich in stearic (saturated) and oleic (**monounsaturated) fatty acids** may be given in limited amounts to boost patients' compliance to a low-fat diet and to prevent their blood lipids from rising to abnormal levels. A bile acid sequestrant (cholestyramine or colestipol) is the first-line drug for control of hypercholesterolemia. Either gemfibrozil or gemfibrozil plus niacin is prescribed to raise high-density lipoprotein (HDL) levels of CAD patients. Approval of two HMG CoA reductase inhibitors, pravastatin and simvastatin, by the FDA gives physicians the additional flexibility of employing a single or a combination drug therapy for optimal control of dyslipidemia. The association of low serum cholesterol level (< 160mg/dl) with increase in noncardiac mortality has prompted health professionals to consider modifying the universal screening and treatment of serum cholesterol in children and young women and to use hypolipidemic drugs in patients judiciously.

L27 ANSWER 10 OF 11 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
ACCESSION NUMBER: 2004-784495 [77] WPIDS
CROSS REFERENCE: 2004-784494 [77]; 2005-111211 [12]
DOC. NO. CPI: C2004-274532
TITLE: Formation of cargo moiety-cochleate, useful for treating inflammation, pain, infection, or fungal infection involves introducing cargo moiety to liposome in presence of solvent followed by precipitation.
DERWENT CLASS: A96 B04 B05 D16
INVENTOR(S): DELMARRE, D; GOULD-FOGERITE, S; KRAUSE-ELSMORE, S L; LU, R; MANNINO, R J
PATENT ASSIGNEE(S): (BIOD-N) BIODELIVERY SCI INT INC; (UYNE-N) UNIV NEW JERSEY MEDICINE & DENTISTRY
COUNTRY COUNT: 108
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004091578	A2	20041028	(200477)*	EN	195

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004091578	A2	WO 2004-US11026	20040409

PRIORITY APPLN. INFO: US 2004-537252P 20040115; US

2003-532755P 20031224

ED 20041203
AN 2004-784495 [77] WPIDS
CR 2004-784494 [77]; 2005-111211 [12]
AB WO2004091578 A UPAB: 20050218

NOVELTY - Formation (F1) of a cargo moiety-cochleate (A1) involves: introducing a cargo moiety to a liposome in the presence of a solvent such that the cargo moiety associates with the liposome; and precipitating the liposome to form a cargo moiety-cochleate.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(a) an article of manufacture comprising packaging material and a lipid contained within the packaging material. The packaging material comprises a label or package insert indicating the use of the lipid for forming cochleates or cochleate compositions;

(b) a composition comprising an anhydrous cochleate; and

(c) formation (F2) of an anhydrous cochleate involving containing a negatively charged lipid, a protonized cargo moiety and a divalent metal cation.

ACTIVITY - Antiinflammatory; Analgesic; Antimicrobial; Fungicide; Antibacterial; Virucide; Antiparasitic; Immunomodulator; Cytostatic; Anorectic; Antidepressant; Vasotropic; Hypotensive; Nootropic; Eating-Disorder-Gen.; Neuroleptic; Tranquilizer; Neuroprotective; Antiparkinsonian; Hemostatic; Anticoagulant; Immunosuppressive; Muscular-Gen.; Neuroprotective; Antianemic; Antithyroid; Antiarthritic; Antirheumatic; Antipsoriatic; Dermatological; Ophthalmological; Antilipemic; CNS-Gen.; Respiratory-Gen.; Osteopathic; Antiarteriosclerotic; Antigout; Antiasthmatic; Auditory; Gastrointestinal-Gen.; Antiulcer.

MECHANISM OF ACTION - None given.

USE - For treating inflammation, pain, infection, fungal infection, bacterial infection, viral infection, parasite disorders, an immune disorder, genetic disorders, degenerative disorders, cancer, proliferative disorders, obesity, depression, hair loss, impotence, hypertension, hypotension, dementia, senile dementia, malnutrition, acute or chronic leukemia or lymphoma, sarcoma, adenoma, carcinomas, epithelial cancer, small cell lung cancer, non-small cell lung cancer, prostate cancer, breast cancer, pancreatic cancer, hepatocellular carcinoma, renal cell carcinoma, biliary cancer, colorectal cancer, ovarian cancer, uterine cancer, melanoma, cervical cancer, testicular cancer, esophageal cancer, gastric cancer, mesothelioma, glioma, glioblastoma, pituitary adenomas, schizophrenia, obsessive compulsive disorder (OCD), bipolar disorder, Alzheimer's disease, Parkinson's disease, cell proliferative disorders, blood coagulation disorders, Dysfibrinogenaemia and hemophilia (A and B), autoimmune disorders, systemic lupus erythematosus, multiple sclerosis, myasthenia gravis, autoimmune hemolytic anemia, autoimmune thrombocytopenia, Grave's disease, allogenic transplant rejection, ankylosing spondylitis, psoriasis, scleroderma, uveitis, eczema, dermatological disorders, hyperlipidemia, **hyperglycemia**, hypercholesterolemia, cystic fibrosis, muscular dystrophy, headache, arthritis, rheumatoid arthritis, osteoarthritis, atherosclerosis, acute gout, acute or chronic soft tissue damage, asthma, chronic rhinosinusitis, allergic fungal sinusitis, sinus mycetoma, non-invasive fungus induced mucositis, non-invasive fungus induced intestinal mucositis, chronic otitis, media, chronic colitis, inflammatory bowel disease, ulcerative colitis or Crohn's disease (all claimed).

ADVANTAGE - The cochleate safely and effectively deliver cargo moieties that are poorly absorbed by the body. The cochleate obtained is

cost-effective and time perspective. The cochleate minimizes the incidence of toxic side effects and/or buildup of cargo moiety in the digestive tract. The cochleate avoids harmful side effects of drugs caused by their

L27 ANSWER 11 OF 11 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
ACCESSION NUMBER: 2003-229216 [22] WPIDS
CROSS REFERENCE: 2004-191208 [18]; 2004-191209 [18]; 2004-191210 [18]; 2004-294860 [27]
DOC. NO. CPI: C2003-058784
TITLE: Orally deliverable pharmaceutical composition useful for treating e.g. headache comprises low water solubility drug and solvent liquid.
DERWENT CLASS: A96 B03 B05
INVENTOR(S): FORBES, J C; GAO, P; HASSAN, F; KARIM, A
PATENT ASSIGNEE(S): (PHAA) PHARMACIA CORP; (FORB-I) FORBES J C; (GAOP-I) GAO P; (HASS-I) HASSAN F; (KARI-I) KARIM A
COUNTRY COUNT: 101
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002083177	A1	20021024	(200322)*	EN	24
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW					
US 2003105141	A1	20030605	(200339)		
NO 2003004629	A	20031210	(200406)		
EP 1379279	A1	20040114	(200410)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
BR 2002008994	A	20040427	(200430)		
AU 2002305175	A1	20021028	(200433)		
CZ 2003002792	A3	20040414	(200435)		
KR 2004018355	A	20040303	(200443)		
JP 2004530669	W	20041007	(200466)		109
CN 1516601	A	20040728	(200469)		
MX 2003009411	A1	20040201	(200473)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2002083177	A1		WO 2002-US11689	20020412
US 2003105141	A1	Provisional	US 2001-284381P	20010417
		Provisional	US 2001-326952P	20011004
			US 2002-119129	20020409
NO 2003004629	A		WO 2002-US11689	20020412
			NO 2003-4629	20031016
EP 1379279	A1		EP 2002-733979	20020412
			WO 2002-US11689	20020412
BR 2002008994	A		BR 2002-8994	20020412
			WO 2002-US11689	20020412
AU 2002305175	A1		AU 2002-305175	20020412
CZ 2003002792	A3		WO 2002-US11689	20020412
			CZ 2003-2792	20020412
KR 2004018355	A		KR 2003-713651	20031017
JP 2004530669	W		JP 2002-580978	20020412

		WO 2002-US11689	20020412
CN 1516601	A	CN 2002-812078	20020412
MX 2003009411	A1	WO 2002-US11689	20020412

PATENT NO	KIND		PATENT NO
EP 1379279	A1	Based on	WO 2002083177
BR 2002008994	A	Based on	WO 2002083177
AU 2002305175	A1	Based on	WO 2002083177
CZ 2003002792	A3	Based on	WO 2002083177
JP 2004530669	W	Based on	WO 2002083177
MX 2003009411	A1	Based on	WO 2002083177

PRIORITY APPLN. INFO: US 2001-326952P 20011004; US 2001-284381P 20010417; US 2002-119129 20020409

ED 20030402

AN 2003-229216 [22] WPIDS

CR 2004-191208 [18]; 2004-191209 [18]; 2004-191210 [18]; 2004-294860 [27]

AB WO 200283177 A UPAB: 20041112

NOVELTY - An orally deliverable pharmaceutical composition (A) comprises a drug (a) (1-75 %) of low water solubility and a solvent liquid (b).

DETAILED DESCRIPTION - An orally deliverable pharmaceutical composition (A) comprises a drug (a) (1-75 %) of low water solubility and a solvent liquid (b). (b) Comprises at least one solvent (c), at least one fatty acid (d) and at least one organic amine (e). A portion of (a) is in dissolved or solubilized form in (b). (d) And (e) are present in total and relative amount such that (A) is finely self-**emulsifiable** in simulated gastric fluid.

ACTIVITY - Antibacterial; Immunosuppressive; Antipyretic; Antiasthmatic; Antiulcer; Hemostatic; Antirheumatic; Antiarthritic; Osteopathic; Dermatological; Hepatotropic; Virucide; Antipsoriatic; Antiseborrheic; Vasotropic; Antithyroid; Antianemic; Neuroprotective; Antiallergic; Opthalmological; Nootropic; Cerebroprotective; Analgesic; Gynecological; Antiinflammatory; Cardiant; Antiarteriosclerotic; Tranquilizer; Vulnerary; Antianginal; Antidiabetic; Cytostatic; Nephrotropic.

MECHANISM OF ACTION - Tumor angiogenesis inhibitor.

No biological data given.

USE - For treating a medical condition or disorder including headache or migraine (claimed). Also useful for treating inflammation and fever, peptic ulcers, gastritis, regional enteritis, ulcerative colitis, diverticulitis or with recurrent history of gastrointestinal lesions, gastrointestinal bleeding, coagulation disorders including anemia, kidney disease, in patients prior to surgery or patient taking anticoagulants, arthritic disorders (e.g. rheumatoid arthritis, spondyloarthropathies, gouty arthritis, osteoarthritis, systemic lupus erythematosus and juvenile arthritis), asthma, bronchitis, menstrual cramps, preterm labor, tendinitis, bursitis, allergic neuritis, cytomegalovirus infectivity, apoptosis including HIV-induced apoptosis, lumbago liver disease including hepatitis, skin-related conditions such as psoriasis, eczema, acne, burns, dermatitis and ultraviolet radiation damage including sunburn and post-operative inflammation including ophthalmic surgery e.g. cataract surgery or refractive surgery, gastrointestinal conditions (e.g. inflammatory bowel disease, Crohn's disease, gastritis and irritable bowel syndrome), treating inflammation in diseases (e.g. periarteritis nodosa, thyroiditis, aplastic anemia, Hodgkin's disease, scleroderma, rheumatic fever, **type I diabetes**, neuromuscular junction disease including myasthenia gravis, white matter disease including multiple sclerosis, sarcoidosis, nephrotic syndrome, Behcet's syndrome, polymyositis, gingivitis, nephritis, hypersensitivity, swelling

occurring after injury (e.g. retinitis, conjunctivitis, retinopathies, uveitis, ocular photophobia and of acute injury to the eye tissue), pulmonary inflammation, e.g. viral infections and cystic fibrosis, in bone and liver disease, pain, fever and inflammation in influenza and other viral infections including common cold, low back and neck pain, dysmenorrhea, toothache, sprains and strains, myositis, neuralgia, synovitis, inflammation-related cardiovascular disorders (e.g. vascular disease, coronary artery disease, aneurysm, vascular rejection, arteriosclerosis, atherosclerosis (e.g. cardiac transplant, myocardial infarction, embolism, stroke, thrombosis including venous thrombosis, angina (e.g. unstable angina, coronary plaque inflammation, bacterial-induced inflammation e.g. chlamydia-induced inflammation, viral induced inflammation, and inflammation associated with surgical procedures e.g. vascular grafting such as coronary artery bypass surgery, revascularization procedures e.g. angioplasty, stent placement, endarterectomy or other invasive procedures, to inhibit tumor angiogenesis, treating neoplasia e.g. metastasis, ophthalmological conditions e.g. corneal graft rejection, ocular neovascularization, retinal neovascularization e.g. injury or infection, diabetic retinopathy, macular degeneration, retrolental fibroplasia and neovascular glaucoma, ulcerative diseases e.g. gastric ulcer, hemangiomas e.g. infantile hemangiomas, angiofibroma of nasopharynx and avascular necrosis of bone, disorders of the female reproductive system e.g. endometriosis, for treating benign, malignant tumors, neoplasia and cancer. The composition has reduced potential for gastrointestinal toxicity and gastrointestinal irritation (upper gastrointestinal ulceration and bleeding), renal side effects (e.g. reduction in renal function leading to fluid retention and exacerbation of hypertension), reduced effect on bleeding times including (inhibition of platelet function and possibly a lessened ability to induce asthma attacks in aspiring-sensitive asthmatic subjects).

ADVANTAGE - (A) Permits a high concentration of the drug and permit rapid adsorption of the drug into the bloodstream through formation of a fine emulsion in the aqueous environment of the gastrointestinal tract and thus provide rapid onset of therapeutic action.

Dwg.0/0

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LAST RELOADED: Apr 15, 2005 (20050415/UP).

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     (FILE 'HOME' ENTERED AT 10:45:15 ON 22 APR 2005)

     FILE 'HCAPLUS' ENTERED AT 10:45:27 ON 22 APR 2005
               E HEIRLER H/AU
```

```
L1             2 S E4

     FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS' ENTERED AT 10:46:14 ON 22

L5        122681 S (LINOLEIC ACID?) OR LINOLEATE? OR "9,12-OCTADECADIENOIC ACID"
L6          2949 S (Ω)(W)"6"(W)FATTY(W)ACID? OR (DOUBLE(W)UNSATURATED(W)TR
L7         85651 S ((A)(W)LINOLENIC(W)ACID?) OR (LINOLENIC ACID?) OR LINOL
L8             1 S (TRIPLE(W)UNSATURATED(W)TRIGLYCERIDE?)
L9         52757 S EICOSAPENTAEN? OR EICOSAPENTAENOIC ACID? OR TIMNODONIC ACID?
L10        60428 S DOCOSAHEXAEN? OR DOCOSAHEXAENOIC ACID? OR "DHA" OR (FISH OIL?
L11         4405 S (SATURATED(W)LONG(W)CHAIN(W)(TRIGLYCERIDE? OR TRIACYLGLYCEROL
L12       152425 S EMULSIFIER OR EMULSIF?
L13        69467 S MONOGLYCERIDE? OR MONOACYLGLYCEROL? OR DIGLYCERIDE? OR DIACYL
L14       421736 S "VITAMIN A" OR RETINOL? OR RETINYL PALMITATE? OR "VITAMIN D"
L15        48454 S CAROTIN? OR (BETA(W)(CAROTIN? OR CAROTENE?)) OR ((B)(W)
L16        27388 S FLAVORING? OR (BUTTER(W)FLAVOR?) OR ROSEMARY? OR (ROSEMARY(W)
L17       130972 S RETINYL PALMITATE? OR "VITAMIN D3" OR CHOLECALCIFEROL? OR "VI
L18       197160 S "VITAMIN B6" OR PICOLINE? OR PYRIDOXINE? OR PYRIDOXAL? OR PYR
L19       404698 S "VITAMIN C" OR ASCORBIC ACID? OR ASCORBYL PALMITATE? OR "VITA
L20      4846639 S ZINC? OR CHROM? OR MANGANESE?
L21        32231 S L2 AND L3
L22          699 S L21 AND L4
L23          204 S L22 AND (L5 OR L6 OR L7 OR L8)
L24           73 S L23 AND (L9 OR L10)
L25            1 S L24 AND L11
L26           14 S L24 AND (L12 OR L13 OR L14 OR L15 OR L16)
L27           11 DUP REM L26 (3 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 11:18:33 ON 22 APR 2005

=> s 124 not 126
             1 DIABETES
             0 MELLITUS
             0 DIABETES(W) MELLITUS
           191 "TYPE"
            18 "TYPES"
           191 "TYPE"
                 ("TYPE" OR "TYPES")
           225 "1"
             1 "DIABETES"
             0 "TYPE 1 DIABETES"
                 ("TYPE"(W)"1"(W)"DIABETES")
           191 "TYPE"
            18 "TYPES"
           191 "TYPE"
                 ("TYPE" OR "TYPES")
           223 "2"
             1 "DIABETES"
             0 "TYPE 2 DIABETES"
                 ("TYPE"(W)"2"(W)"DIABETES")
           191 "TYPE"
            18 "TYPES"
           191 "TYPE"
                 ("TYPE" OR "TYPES")
            21 "I"
             1 "DIABETES"
             0 "TYPE I DIABETES"
                 ("TYPE"(W)"I"(W)"DIABETES")
           191 "TYPE"
            18 "TYPES"
           191 "TYPE"
```

```
    ("TYPE" OR "TYPES")
 2 "II"
 1 "DIABETES"



 0 DIABETES(W) INSIPIDUS
 0 GLUCOSE
 0 INTOLERAN?
 0 GLUCOSE(W) INTOLERAN?
 0 HYPERGLYCEM?
 0 IMPAIRED
 0 GLUCOSE
 1 TOLERAN?
 0 IMPAIRED GLUCOSE TOLERAN?
    (IMPAIRED(W)GLUCOSE(W)TOLERAN?)
 1 MEDIUM
 4 MEDIA
 5 MEDIUM
    (MEDIUM OR MEDIA)
 0 CHAIN
 0 TRIGLYCERIDE?
 0 MEDIUM(W) CHAIN(W) TRIGLYCERIDE?
 0 TRIACYLGLYCEROL?
 0 TRIGLYCERIDE?
 0 "MCT"
 0 "MCTS"
 0 CAPRIC
 6 ACID?
 0 CAPRIC ACID?
    (CAPRIC(W)ACID?)
 0 CAPRYLIC
 6 ACID?
 0 CAPRYLIC ACID?
    (CAPRYLIC(W)ACID?)
 0 DECANOIC
 6 ACID?
 0 DECANOIC ACID?
    (DECANOIC(W)ACID?)
 0 OCTANOATE?
 0 OCTANOIC
 6 ACID?
 0 OCTANOIC ACID?
    (OCTANOIC(W)ACID?)
 0 OLEIC
 6 ACID?
 0 OLEIC ACID?
    (OLEIC(W)ACID?)
51 "9"
 0 "OCTADECENOIC"
 6 "ACID"
 1 "ACIDS"
 6 "ACID"
    ("ACID" OR "ACIDS")
 0 "9-OCTADECENOIC ACID"
    ("9"(W)"OCTADECENOIC"(W)"ACID")
 0 OLEATE?
 0 OLIVE
14 OIL?
 0 OLIVE OIL?
    (OLIVE(W)OIL?)
 0 RAPE?
14 OIL?
```

```
 0 RAPE? OIL?
     (RAPE?(W)OIL?)
 0 CANOLA



 0 FATTY
 6 ACID?
 0 MONOUNSATURATED(W) FATTY(W) ACID?
 0 MONOENE?
 0 "MUFA"
 0 "MUFAS"
 0 LINOLEIC
 6 ACID?
 0 LINOLEIC ACID?
     (LINOLEIC(W)ACID?)
 0 LINOLEATE?
51 "9"
33 "12"
 0 "OCTADECADIENOIC"
 6 "ACID"
 1 "ACIDS"
 6 "ACID"
     ("ACID" OR "ACIDS")
 0 "9,12-OCTADECADIENOIC ACID"
     ("9"(W)"12"(W)"OCTADECADIENOIC"(W)"ACID")
 0 LINOELAIDIC
 6 ACID?
 0 LINOELAIDIC ACID?
     (LINOELAIDIC(W)ACID?)
 0 SUNFLOWER
14 OIL?
 0 SUNFLOWER OIL?
     (SUNFLOWER(W)OIL?)
 0 RAPE?
14 OIL?
 0 RAPE? OIL?
     (RAPE?(W)OIL?)
 0 OMEGA
76 "6"
 0 FATTY
 6 ACID?
 0 OMEGA(W)"6"(W) FATTY(W) ACID?
 0 OMEGA
76 "6"
 0 FATTY
 6 ACID?
 0 (Ω)(W)"6"(W) FATTY(W) ACID?
 0 DOUBLE
 0 UNSATURATED
 0 TRIGLYCERIDE?
 0 DOUBLE(W) UNSATURATED(W) TRIGLYCERIDE?
 0 ALPHA
 0 LINOLENIC
 6 ACID?
 0 (A)(W) LINOLENIC(W) ACID?
 0 LINOLENIC
 6 ACID?
 0 LINOLENIC ACID?
     (LINOLENIC(W)ACID?)
 0 LINOLENATE?
51 "9"
33 "12"
```

```
 18 "15"
  0 "OCTADECATRIENOIC"
  6 "ACID"


      ("9"(W)"12"(W)"15"(W)"OCTADECATRIENOIC"(W)"ACID")
  0 RAPE?
 14 OIL?
  0 RAPE? OIL?
      (RAPE?(W)OIL?)
  0 LINSEED
 14 OIL?
  0 LINSEED OIL?
      (LINSEED(W)OIL?)
  0 OMEGA
195 "3"
  0 FATTY
  6 ACID?
  0 OMEGA(W)"3"(W) FATTY(W) ACID?
  0 OMEGA
195 "3"
  0 FATTY
  6 ACID?
  0 (Ω)(W)"3"(W) FATTY(W) ACID?
  3 TRIPLE
  0 UNSATURATED
  0 TRIGLYCERIDE?
  0 (TRIPLE(W) UNSATURATED(W) TRIGLYCERIDE?)
  0 EICOSAPENTAEN?
  0 EICOSAPENTAENOIC
  6 ACID?
  0 EICOSAPENTAENOIC ACID?
      (EICOSAPENTAENOIC(W)ACID?)
  0 TIMNODONIC
  6 ACID?
  0 TIMNODONIC ACID?
      (TIMNODONIC(W)ACID?)
  7 "EPA"
  0 DOCOSAHEXAEN?
  0 DOCOSAHEXAENOIC
  6 ACID?
  0 DOCOSAHEXAENOIC ACID?
      (DOCOSAHEXAENOIC(W)ACID?)
  0 "DHA"
  5 FISH
 14 OIL?
  0 FISH OIL?
      (FISH(W)OIL?)
  0 SHELLFISH
  0 TUNA
  0 MACKEREL
  0 SALMON
  0 MENHADEN
  0 MENHADIN
  0 ANCHOVY
  0 HERRING
  0 TROUT
  0 SARDINE
 14 OIL?
  0 (SHELLFISH OR TUNA OR MACKEREL OR SALMON OR MENHADEN OR MENHADIN
     OR ANCHOVY OR HERRING OR TROUT OR SARDINE)(W) OIL?
  1 DIABETES
```

```
  0 MELLITUS
  0 DIABETES(W) MELLITUS
191 "TYPE"



  1 "DIABETES"
  0 "TYPE 1 DIABETES"
      ("TYPE"(W)"1"(W)"DIABETES")
191 "TYPE"
 18 "TYPES"
191 "TYPE"
      ("TYPE" OR "TYPES")
223 "2"
  1 "DIABETES"
  0 "TYPE 2 DIABETES"
      ("TYPE"(W)"2"(W)"DIABETES")
191 "TYPE"
 18 "TYPES"
191 "TYPE"
      ("TYPE" OR "TYPES")
 21 "I"
  1 "DIABETES"
  0 "TYPE I DIABETES"
      ("TYPE"(W)"I"(W)"DIABETES")
191 "TYPE"
 18 "TYPES"
191 "TYPE"
      ("TYPE" OR "TYPES")
  2 "II"
  1 "DIABETES"
  0 "TYPE II DIABETES"
      ("TYPE"(W)"II"(W)"DIABETES")
  1 DIABETES
  0 INSIPIDUS
  0 DIABETES(W) INSIPIDUS
  0 GLUCOSE
  0 INTOLERAN?
  0 GLUCOSE(W) INTOLERAN?
  0 HYPERGLYCEM?
  0 IMPAIRED
  0 GLUCOSE
  1 TOLERAN?
  0 IMPAIRED GLUCOSE TOLERAN?
      (IMPAIRED(W)GLUCOSE(W)TOLERAN?)
  1 MEDIUM
  4 MEDIA
  5 MEDIUM
      (MEDIUM OR MEDIA)
  0 CHAIN
  0 TRIGLYCERIDE?
  0 MEDIUM(W) CHAIN(W) TRIGLYCERIDE?
  0 TRIACYLGLYCEROL?
  0 TRIGLYCERIDE?
  0 "MCT"
  0 "MCTS"
  0 CAPRIC
  6 ACID?
  0 CAPRIC ACID?
      (CAPRIC(W)ACID?)
  0 CAPRYLIC
  6 ACID?
  0 CAPRYLIC ACID?
```

```
     (CAPRYLIC(W)ACID?)
 0 DECANOIC
 6 ACID?
 0 OCTANOIC ACID?
     (OCTANOIC(W)ACID?)
 0 OLEIC
 6 ACID?
 0 OLEIC ACID?
     (OLEIC(W)ACID?)
51 "9"
 0 "OCTADECENOIC"
 6 "ACID"
 1 "ACIDS"
 6 "ACID"
     ("ACID" OR "ACIDS")
 0 "9-OCTADECENOIC ACID"
     ("9"(W)"OCTADECENOIC"(W)"ACID")
 0 OLEATE?
 0 OLIVE
14 OIL?
 0 OLIVE OIL?
     (OLIVE(W)OIL?)
 0 RAPE?
14 OIL?
 0 RAPE? OIL?
     (RAPE?(W)OIL?)
 0 CANOLA
14 OIL?
 0 CANOLA OIL?
     (CANOLA(W)OIL?)
 0 MONOUNSATURATED
 0 FATTY
 6 ACID?
 0 MONOUNSATURATED(W) FATTY(W) ACID?
 0 MONOENE?
 0 "MUFA"
 0 "MUFAS"
 0 LINOLEIC
 6 ACID?
 0 LINOLEIC ACID?
     (LINOLEIC(W)ACID?)
 0 LINOLEATE?
51 "9"
33 "12"
 0 "OCTADECADIENOIC"
 6 "ACID"
 1 "ACIDS"
 6 "ACID"
     ("ACID" OR "ACIDS")
 0 "9,12-OCTADECADIENOIC ACID"
     ("9"(W)"12"(W)"OCTADECADIENOIC"(W)"ACID")
 0 LINOELAIDIC
 6 ACID?
 0 LINOELAIDIC ACID?
     (LINOELAIDIC(W)ACID?)
 0 SUNFLOWER
14 OIL?
 0 SUNFLOWER OIL?
     (SUNFLOWER(W)OIL?)
```

```
  0 RAPE?
 14 OIL?
  0 RAPE? OIL?


  6 ACID?
  0 OMEGA(W)"6"(W) FATTY(W) ACID?
  0 OMEGA
 76 "6"
  0 FATTY
  6 ACID?
  0 (Ω)(W)"6"(W) FATTY(W) ACID?
  0 DOUBLE
  0 UNSATURATED
  0 TRIGLYCERIDE?
  0 DOUBLE(W) UNSATURATED(W) TRIGLYCERIDE?
  0 ALPHA
  0 LINOLENIC
  6 ACID?
  0 (A)(W) LINOLENIC(W) ACID?
  0 LINOLENIC
  6 ACID?
  0 LINOLENIC ACID?
      (LINOLENIC(W)ACID?)
  0 LINOLENATE?
 51 "9"
 33 "12"
 18 "15"
  0 "OCTADECATRIENOIC"
  6 "ACID"
  1 "ACIDS"
  6 "ACID"
      ("ACID" OR "ACIDS")
  0 "9,12,15-OCTADECATRIENOIC ACID"
      ("9"(W)"12"(W)"15"(W)"OCTADECATRIENOIC"(W)"ACID")
  0 RAPE?
 14 OIL?
  0 RAPE? OIL?
      (RAPE?(W)OIL?)
  0 LINSEED
 14 OIL?
  0 LINSEED OIL?
      (LINSEED(W)OIL?)
  0 OMEGA
195 "3"
  0 FATTY
  6 ACID?
  0 OMEGA(W)"3"(W) FATTY(W) ACID?
  0 OMEGA
195 "3"
  0 FATTY
  6 ACID?
  0 (Ω)(W)"3"(W) FATTY(W) ACID?
  3 TRIPLE
  0 UNSATURATED
  0 TRIGLYCERIDE?
  0 (TRIPLE(W) UNSATURATED(W) TRIGLYCERIDE?)
  0 EICOSAPENTAEN?
  0 EICOSAPENTAENOIC
  6 ACID?
  0 EICOSAPENTAENOIC ACID?
      (EICOSAPENTAENOIC(W)ACID?)
```

```
  0 TIMNODONIC
  6 ACID?
  0 TIMNODONIC ACID?

  6 ACID?
  0 DOCOSAHEXAENOIC ACID?
      (DOCOSAHEXAENOIC(W)ACID?)
  0 "DHA"
  5 FISH
 14 OIL?
  0 FISH OIL?
      (FISH(W)OIL?)
  0 SHELLFISH
  0 TUNA
  0 MACKEREL
  0 SALMON
  0 MENHADEN
  0 MENHADIN
  0 ANCHOVY
  0 HERRING
  0 TROUT
  0 SARDINE
 14 OIL?
  0 (SHELLFISH OR TUNA OR MACKEREL OR SALMON OR MENHADEN OR MENHADIN
     OR ANCHOVY OR HERRING OR TROUT OR SARDINE)(W) OIL?
  0 EMULSIFIER
  0 EMULSIF?
  0 MONOGLYCERIDE?
  0 MONOACYLGLYCEROL?
  0 DIGLYCERIDE?
  0 DIACYLGLYCEROL?
  0 "VITAMIN"
227 "A"
  0 "VITAMIN A"
      ("VITAMIN"(W)"A")
  0 RETINOL?
  0 RETINYL
  0 PALMITATE?
  0 RETINYL PALMITATE?
      (RETINYL(W)PALMITATE?)
  0 "VITAMIN"
 27 "D"
  0 "VITAMIN D"
      ("VITAMIN"(W)"D")
  0 CHOLECALCIFEROL?
  0 "VITAMIN"
 81 "E"
  0 "VITAMIN E"
      ("VITAMIN"(W)"E")
  0 TOCOPHEROL?
  0 TOCOTRIENOL?
  0 TOCOPHEROL
  0 ACETATE?
  0 TOCOPHEROL ACETATE?
      (TOCOPHEROL(W)ACETATE?)
  0 "VITAMIN"
 17 "C"
  0 "VITAMIN C"
      ("VITAMIN"(W)"C")
  0 ASCORBYL
  0 PALMITATE?
```

```
              0 ASCORBYL PALMITATE?
                (ASCORBYL(W)PALMITATE?)
              0 CAROTIN?



              0 BETA
              0 CAROTIN?
              0 CAROTENE?
              0 (B)(W) (CAROTIN? OR CAROTENE?)
              0 BELLACAROTIN?
              0 CAROTABEN
              0 PROVATENE?
              0 SOLATENE?
              0 VETORON?
              0 FLAVORING?
              0 BUTTER
              0 FLAVOR?
              0 BUTTER(W) FLAVOR?
              0 ROSEMARY?
              0 ROSEMARY
             57 EXTRACT?
              0 ROSEMARY(W) EXTRACT?
              0 ROSMARINUS?
              0 ROSMARINUS
             57 EXTRACT?
              0 ROSMARINUS(W) EXTRACT?
L28           0 L24 NOT L26

=> file medline biosis hcaplus embase wpids
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                     ENTRY    SESSION
FULL ESTIMATED COST                                   0.42     173.97

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)      SINCE FILE      TOTAL
                                                     ENTRY    SESSION
CA SUBSCRIBER PRICE                                   0.00      -4.38

FILE 'MEDLINE' ENTERED AT 11:22:48 ON 22 APR 2005

FILE 'BIOSIS' ENTERED AT 11:22:48 ON 22 APR 2005
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FILE 'WPIDS' ENTERED AT 11:22:48 ON 22 APR 2005
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=> s l24 not l26
L29          59 L24 NOT L26

=> dup rem l29
PROCESSING COMPLETED FOR L29
L30           40 DUP REM L29 (19 DUPLICATES REMOVED)
                 ANSWERS '1-9' FROM FILE MEDLINE
                 ANSWERS '10-12' FROM FILE BIOSIS
                 ANSWERS '13-27' FROM FILE HCAPLUS
```

ANSWERS '28-39' FROM FILE EMBASE
ANSWER '40' FROM FILE WPIDS

DOCUMENT NUMBER: PubMed ID: 15624100
TITLE: Acute effects of **monounsaturated fatty acids** with and without **omega-3 fatty acids** on vascular reactivity in individuals with **type 2 diabetes**.
AUTHOR: West S G; Hecker K D; Mustad V A; Nicholson S; Schoemer S L; Wagner P; Hinderliter A L; Ulbrecht J; Ruey P; Kris-Etherton P M
CORPORATE SOURCE: Department of Biobehavioral Health, The Pennsylvania State University, University Park, PA, USA.
CONTRACT NUMBER: M01RR10732 (NCRR)
SOURCE: Diabetologia, (2005 Jan) 48 (1) 113-22. Electronic Publication: 2004-12-29.
Journal code: 0006777. ISSN: 0012-186X.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20050125
Last Updated on STN: 20050301

ED Entered STN: 20050125
Last Updated on STN: 20050301

AB AIMS/HYPOTHESIS: We examined the acute postprandial effects of meals containing unsaturated fatty acids on flow-mediated dilation (FMD) of the brachial artery and **triacylglycerols** in individuals with **type 2 diabetes**. We hypothesised that consumption of **omega-3 fatty acids** would enhance vascular function. Saturated fat reduces FMD for several hours, but there is inconsistent evidence about whether foods containing unsaturated fats impair FMD acutely. Little is known about the acute effects of **omega-3 fatty acids** on vascular reactivity. METHODS: We measured FMD before and 4 h after 3 test meals (50 g fat, 2,615 kJ) in 18 healthy adults with **type 2 diabetes**. The **monounsaturated fatty acids** (**MUFA**) meal contained 50 g fat from high oleic safflower and **canola oils**. Two additional meals were prepared by replacing 7% to 8% of **MUFA** with **docosahexaenoic acid** and **eicosapentaenoic acid** from **sardine oil** or **alpha-linolenic acid** from **canola oil**. RESULTS: In the sample as a whole, FMD was increased 17% at 4 h vs. the fasting baseline. After the **MUFA** meal, subjects with the largest increases in **triacylglycerols** had the largest FMD decreases. The opposite pattern was observed after meals containing **docosahexaenoic acid** and **eicosapentaenoic acid** or **alpha-linolenic acid**. In subjects with high fasting **triacylglycerols**, meals containing 3 to 5 g of **omega-3 fatty acids** increased FMD by 50% to 80% and **MUFA** alone had no significant effects on FMD. CONCLUSIONS/INTERPRETATION: Endothelium-dependent vasodilation was not impaired 4 h after meals containing predominantly unsaturated fatty acids. The fatty acid composition of the meal and the metabolic status of the individual determine the vascular effects of a high-fat meal.

L30 ANSWER 2 OF 40 MEDLINE on STN DUPLICATE 3

ACCESSION NUMBER: 2002450161 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12210759
TITLE: Effects of alpha lipoic acid, ascorbic acid-6-palmitate

Ersan Yasemin
CORPORATE SOURCE: Department of Biology, Faculty of Science, Firat University, 23169-Elazig, Turkey.. oyilmaz@firat.edu.tr
SOURCE: Journal of cellular biochemistry, (2002) 86 (3) 530-9.
Journal code: 8205768. ISSN: 0730-2312.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200303
ENTRY DATE: Entered STN: 20020906
Last Updated on STN: 20030313
Entered Medline: 20030312

ED Entered STN: 20020906
Last Updated on STN: 20030313
Entered Medline: 20030312

AB In this research, it has been aimed to evaluate the improvement effects of alpha lipoic acid (ALA), ascorbic acid-6-palmitate (AA6P), **fish oil** (FO), and their combination (COM) on some biochemical properties in erythrocytes of streptozotocin (STZ)-induced diabetic male rats. According to experimental results, glutathione (GSH) level in erythrocytes decreased in diabetes ($P < 0.01$), D + ALA, and D + AA6P groups ($P < 0.001$). Malonaldehyde (MA) level increased in diabetes ($P < 0.05$), D + FO, and D + COM groups ($P < 0.001$), but its level in D + AA6P and D + ALA groups was lower in diabetes group ($P < 0.01$). Total lipid level in diabetes and diabetes plus antioxidant administered groups were higher than control. Total cholesterol level was high in diabetes and D + ALA groups ($P < 0.05$), but its level reduced in D + FO compared to control and diabetes groups, $P < 0.05$, < 0.001, respectively. Total **triglyceride** (TTG) level was high in the D + ALA ($P < 0.05$) and D + COM ($P < 0.001$) groups. In contrast, TTG level in blood of diabetes group was higher than diabetes plus antioxidant and FO administered groups ($P < 0.001$). According to gas chromatography analysis results, while the palmitic acid raised in diabetes group ($P < 0.05$), stearic acid in D + FO, D + ALA, and diabetes groups was lower than control ($P < 0.05$), **oleic acid** reduced in D + COM and D + FO groups, but its level raised in D + AA6P and D + ALA groups ($P < 0.01$). As the **linoleic acid** (LA) elevated in ALA + D, D + AA6P, and diabetes groups, **linolenic acid** level in diabetes, D + AA6P, and D + FO groups was lower than control ($P < 0.001$). Arachidonic acid (AA) decreased in D + ALA, D+ AA6P, and diabetes groups ($P < 0.01$), but its level in D + COM and D + FO was higher than control ($P < 0.05$). **Docosahexaenoic acid (DHA)** increased in D + AA6P and D + COM ($P < 0.05$). While the total saturated fatty acid level raised in diabetes group, its level reduced in D + ALA and D + FO groups ($P < 0.05$). In contrast, total unsaturated fatty acid level in D + ALA and D + FO groups was higher than control ($P < 0.05$). In conclusion, present data have confirmed that the combination of the ALA, AA6P, and FO have improvement effects on the recycling of GSSG to reduced GSH in erythrocytes of diabetic rats, and in addition to this, oxidative stress was suppressed by ALA and AA6P, and unsaturated fatty acid degree was raised by the effects of ALA and FO.

L30 ANSWER 3 OF 40 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2002063384 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11789299

TITLE: The effect of **omega-3 fatty acids** on risk factors for cardiovascular diseases.

AUTHOR: [illegible]

DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: Hebrew
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200202
ENTRY DATE: Entered STN: 20020125
Last Updated on STN: 20020216
Entered Medline: 20020215

ED Entered STN: 20020125
Last Updated on STN: 20020216
Entered Medline: 20020215

AB Cardiovascular disease (CVD) is associated with dyslipidemia and frequently with insulin resistance, both of which are in general no alleviated by antilipidemic drugs. Our objective was to examine whether a dietary supplement containing **omega-3 fatty acids** (n-3 FA) can reduce the levels of serum lipids, fasting insulin and glucose in documented CVD patients treated by statins or bezafibrates. In a double-blind placebo-controlled trial of parallel design, 52 patients, age 69.2 years +/- 3.6 treated by antilipidemic drugs, were randomly assigned to receive daily 7 gr of a dietary concentrated supplement containing 67% n-3 FA (185 mg **EPA** and 465 mg/g **DHA**) in a form of spread (Yamega Ltd, Israel) or **olive oil** spread (placebo) and recommended to reduce the consumption of **omega-6 fatty acids** for 12 weeks. The average values +/- SD before and after dietary supplementations were compared. RESULTS: 44 patients (23 in the n-3 FA group) completed the study. In the n-3FA group we observed a significant decrease ($p < 0.05$) of total cholesterol (12.2%). LDL-cholesterol (16.8%), **triglycerides** (36.1%), insulin in hyperinsulinemic subjects (> 20 microunits/ml) (34.9%), and no significant changes in HDL-cholesterol and glucose. No **hyperglycemia** was detected. In the **olive oil** group we observed a significant decrease ($p < 0.05$) in the LDL-cholesterol values of 15.5% and no significant changes in the other parameters. No side effects were reported during the study in any of the participants. Our findings demonstrate that the incorporation of the dietary supplement containing **EPA** and **DHA omega-3 fatty acids** reduces significantly the above risk factors for CVD.

L30 ANSWER 4 OF 40 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 1999221278 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10206437
TITLE: Effects of dietary fatty acids on lipid metabolism in streptozotocin-induced diabetic rats.
AUTHOR: Giron M D; Sanchez F; Hortelano P; Periago J L; Suarez M D
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, School of Pharmacy, Granada, Spain.
SOURCE: Metabolism: clinical and experimental, (1999 Apr) 48 (4) 455-60.
Journal code: 0375267. ISSN: 0026-0495.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 19990504

Last Updated on STN: 19990504
Entered Medline: 19990422

ED Entered STN: 19990504

control and diabetic rats fed a basal diet supplemented with 5% (by weight) **olive oil** (OO), **sunflower oil** (SO), or **fish oil** (FO), respectively. Plasma glucose, cholesterol, triacylglyceride, and phospholipid levels were also measured. An increase in plasma and liver microsome **oleic acid** and a decrease in arachidonic acid were found in diabetes. In the liver, **docosahexaenoic acid** levels were higher in diabetic versus control rats. Diabetes increased liver delta9-desaturase in OO-fed rats and did not modify delta6-desaturase activity in OO- or SO-fed rats. Both enzymatic activities were decreased in diabetic rats fed the FO diet. As a main conclusion, it appears that diet-induced alterations in membrane composition provide a mechanism for improving the diabetic condition in animals and overcoming the effect of insulin deficiency on desaturase activities. Plasma cholesterol was not modified either by diabetes or by diet. In diabetes, OO-fed rats showed the lowest levels of **triglycerides**. Plasma phospholipids were significantly higher in OO-fed versus FO-fed rats. These findings suggest that OO contributes to a better control of the hypertriglyceridemia accompanying diabetes as compared with the other two diets in this rat model.

L30 ANSWER 5 OF 40 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 97180541 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9028717
TITLE: Effect of **omega 3 fatty acid** on plasma lipids, cholesterol and lipoprotein fatty acid content in NIDDM patients.
AUTHOR: Goh Y K; Jumpsen J A; Ryan E A; Clandinin M T
CORPORATE SOURCE: Nutrition and Metabolism Research Group, University of Alberta, Edmonton, Canada.
SOURCE: Diabetologia, (1997 Jan) 40 (1) 45-52.
Journal code: 0006777. ISSN: 0012-186X.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 19970721
Last Updated on STN: 19970721
Entered Medline: 19970708
ED Entered STN: 19970721
Last Updated on STN: 19970721
Entered Medline: 19970708
AB This study was conducted to examine the effect of **omega 3 fatty acid** supplementation on plasma lipid, cholesterol and lipoprotein fatty acid content of non-insulin-dependent diabetic individuals consuming a higher (0.65, n = 10) or lower (0.44, n = 18) ratio of dietary polyunsaturated to saturated fatty acid (P/S). The participants were initially given an **olive oil** supplement (placebo) equivalent to 35 mg of 18:1. kg body weight-1.day-1 for 3 months. This was followed by two omega 3 supplement periods in a randomized crossover. In these 3-month periods, participants were given a **linseed oil** supplement equivalent to 35 mg of 18:3 omega 3.kg body weight-1.day-1 or a **fish oil** supplement equivalent to 35 mg of 20:5 omega 3 + 22:6 omega 3.kg body weight-1.

day-1. At the end of each supplement period, a blood sample was drawn from each participant for lipid, lipoprotein, insulin, glucagon and C-peptide analyses. At the end of each 3 month period a 7 day diet...

Linolenic acid supplementation had no effect on plasma **triacylglycerol** level, but it increased 18:3 omega 3 content of lipoprotein cholesterol ester fractions ($p < 0.05$). A slight increase in 20:5 omega 3, but not 22:6 omega 3, content was noted in lipoprotein lipid classes as a result of 18:3 omega 3 supplementation. LDL and HDL cholesterol, insulin, glucagon and C-peptide levels were not affected by either omega 3 supplement. It is concluded that a modest intake of **omega 3 fatty acids**, such as could be obtained from consuming fish regularly, will reduce plasma **triglyceride** level without affecting LDL or HDL cholesterol levels.

L30 ANSWER 6 OF 40 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 96311092 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8732710
TITLE: A comparison of the effects of n-3 fatty acids from **linseed oil** and **fish oil** in well-controlled **type II diabetes**.
AUTHOR: McManus R M; Jumpson J; Finegood D T; Clandinin M T; Ryan E A
CORPORATE SOURCE: Department of Medicine, University of Alberta, Edmonton, Canada.
SOURCE: Diabetes care, (1996 May) 19 (5) 463-7.
Journal code: 7805975. ISSN: 0149-5992.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199609
ENTRY DATE: Entered STN: 19961008
Last Updated on STN: 19961008
Entered Medline: 19960920
ED Entered STN: 19961008
Last Updated on STN: 19961008
Entered Medline: 19960920
AB OBJECTIVE--Supplementation of type II diabetic diets with n-3 fatty acids (FAs) from **fish oil** (FO) has been associated with lowered **triglyceride** and VLDL levels, although reports of impaired glycemic control have limited their use. Effects of n-3FAs from nonmarine sources are less well documented. Therefore, an investigation comparing the effects of **linseed oil** (LO) with FO supplementation was undertaken in subjects with **type II diabetes**. RESEARCH DESIGN AND METHODS--Eleven subjects with **type II diabetes** were given supplements with LO and FO for 3 months each in a randomized double-blind crossover fashion after 3 months of **olive oil** placebo. Oils were given as 35 mg FA.kg body wt-1.day-1. After each 3-month period, fasting glucose and insulin levels, HbA1c, lipid profiles, insulin sensitivity (SI), glucose effectiveness (SG), and acute insulin response to glucose (AIRG) were evaluated. RESULTS--HbA1c and lipid values were within the normal range at randomization. Repeated measures analysis of variance testing found no significant differences in weight; fasting glucose and insulin levels; HbA1c; total, LDL, and HDL cholesterol levels; SI; SG; or AIRG with either active oil. FO was associated with significant

reductions in **triglycerides** and a trend toward decreased SI. CONCLUSIONS--In a population with well-controlled **type** II diabetes, 3 months of EO but not IO resulted in

L30 ANSWER 7 OF 40 MEDLINE on STN DUPLICATE 9
ACCESSION NUMBER: 92097857 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1756905
TITLE: Decreased incorporation of long-chain fatty acids into erythrocyte phospholipids of STZ-D rats.
AUTHOR: Dang A Q; Faas F H; Jethmalani S M; Carter W J
CORPORATE SOURCE: John L. McClellan Memorial Veterans Hospital, Little Rock, AR 72205.
SOURCE: Diabetes, (1991 Dec) 40 (12) 1645-51.
Journal code: 0372763. ISSN: 0012-1797.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199202
ENTRY DATE: Entered STN: 19920223
Last Updated on STN: 19920223
Entered Medline: 19920203
ED Entered STN: 19920223
Last Updated on STN: 19920223
Entered Medline: 19920203
AB We studied the mechanisms for the altered fatty acid composition in erythrocytes (RBCs) derived from streptozocin-induced diabetic (STZ-D) rats. After 3-wk duration of diabetes, blood glucose, plasma **triglyceride**, and plasma free-fatty acid levels were all significantly increased. In the diabetic platelet-poor plasma (PPP), the most significant increases in free fatty acids were stearate, **linoleate**, eicosatrienoate (n-6), and **docosahexaenoate** (n-3). Fatty acid composition of RBC phospholipids was also altered, with significant decreases in arachidonate, docosatetraenoate (n-6), and docosapentaenoate (n-6) and increases in **linoleate** and **docosahexaenoate**. Insulin treatment of the diabetic rats resulted in normalization of docosapentaenoate, arachidonate, and **linoleate** levels in RBC phospholipids but not of **docosahexaenoate** or docosatetraenoate levels. The incorporation of [5,6,8,9,11,12,14,15-3H]arachidonate into diabetic RBC phospholipids was significantly decreased compared with the corresponding control RBC, regardless of the incubation medium used, which was the PPP derived either from the control or diabetic rats. Therefore, the decreased incorporation of [5,6,8,9,11,12,14,15-3H]arachidonate into diabetic RBC phospholipids was independent of the altered lipid composition of the PPP incubation media. Furthermore, the decreased incorporation was not specific for arachidonate, because the incorporation of other long-chain fatty acids such as [9,10-3H]**oleate**, [1-14C]palmitate, [2-14C]eicosatrienoate (n-6), and [1-14C]**linoleate** into RBC phospholipids was also comparably decreased. More important, the decreased fatty acid incorporations were reversed by insulin treatment of the diabetic rat.(ABSTRACT TRUNCATED AT 250 WORDS)

L30 ANSWER 8 OF 40 MEDLINE on STN DUPLICATE 10
ACCESSION NUMBER: 91155740 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2000036
TITLE: Comparison of diets supplemented with **fish oil** or **olive oil** on plasma lipoproteins in insulin-dependent diabetics.
AUTHOR: Mori T A; Vandongen R; Masarei J R; Rouse I L; Dunbar D

CORPORATE SOURCE: University Department of Medicine, Royal Perth Hospital, Western Australia.
SOURCE: [illegible]

Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199104
ENTRY DATE: Entered STN: 19910428
Last Updated on STN: 19950206
Entered Medline: 19910411

ED Entered STN: 19910428
Last Updated on STN: 19950206
Entered Medline: 19910411

AB This study was designed to compare changes in high-density lipoprotein (HDL)- and low-density lipoprotein (LDL)-cholesterol in normolipidaemic male insulin-dependent diabetics (IDD) following dietary supplementation with either the **fish oil** concentrate Max **EPA** or **olive oil**. The contribution of the small quantity of cholesterol in Max **EPA** to these changes was also examined. Twenty-seven subjects were matched in groups of three and randomly allocated to one of three treatment groups of nine subjects each. Subjects were given 15 1-g capsules of oil daily for 3 weeks, consisting of either Max **EPA**, **olive oil**, or **olive oil** to which was added the same amount of cholesterol as contained in Max **EPA**, respectively. There was a significant increase in **eicosapentaenoic acid**, and a decrease in arachidonic acid, in the platelet membrane phospholipids of subjects taking Max **EPA**. In this group, there was an approximately 30% increase in serum HDL2-cholesterol (0.59 +/- 0.07 to 0.77 +/- 0.11 mmol/L, mean +/- SEM; P less than .01) and a corresponding decrease in HDL3-cholesterol (0.79 +/- 0.03 to 0.71 +/- 0.03 mmol/L; P less than .05). Although total and LDL-cholesterol concentrations were also higher after Max **EPA**, the changes were not significant. **Triglycerides** were significantly decreased by Max **EPA**. There were no significant changes in lipids in the groups given **olive oil**. These results show that compared with **olive oil**, dietary supplementation with Max **EPA** substantially increases HDL2-cholesterol in insulin-dependent diabetics. This is most likely due to a selective effect of **omega 3 fatty acids**.(ABSTRACT TRUNCATED AT 250 WORDS)

L30 ANSWER 9 OF 40 MEDLINE on STN
ACCESSION NUMBER: 89039284 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3185290
TITLE: Altered fatty acid composition in the plasma, platelets, and aorta of the streptozotocin-induced diabetic rat.
AUTHOR: Dang A Q; Faas F H; Lee J A; Carter W J
CORPORATE SOURCE: John L. McClellan Memorial Veterans Hospital, Little Rock, AR.
SOURCE: Metabolism: clinical and experimental, (1988 Nov) 37 (11) 1065-72.
Journal code: 0375267. ISSN: 0026-0495.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198812
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308

Entered Medline: 19881216
ED Entered STN: 19900308
Last Updated on STN: 19900308

communication, we describe experiments that indicate that in the short-term streptozotocin diabetic rat (2 to 3 weeks), the fatty acid composition of plasma and red blood cell lipids was altered but remained unchanged in platelet and aorta phospholipids. The altered fatty acid composition of the diabetic red blood cells and plasma cholesterol esters and phospholipids was similar to that previously found in the diabetic liver. However, in long-term diabetes (6 weeks), the phospholipid fatty acid composition of the platelet and aorta became significantly altered. Thus, in the 6-week diabetic platelet, there were increases of **linoleate**, dihomo-gamma-**linolenate**, docosapentaenoate (C22:5n-3), and **docosahexaenoate**, and decreases of **oleate**, arachidonate, and docosatetraenoate. In the aorta, there were increases of **linoleate**, **eicosapentaenoate**, and **docosahexaenoate**, and decreases of arachidonate, docosatetraenoate, and docosapentaenoate (C22:5n-6). Results from these experiments indicate that the fatty acid composition of plasma and red blood cell lipids was altered in short-term diabetes (2 to 3 weeks), but that of platelet and aorta phospholipids was not changed until more prolonged diabetes was present. Insulin treatment of the diabetic rat increased the levels of palmitoleate and **oleate** and decreased the levels of **linoleate** in platelet and aorta lipids from insulin-treated diabetic rats, suggesting an overcorrection of diminished delta 9 and delta 6 fatty acid desaturation as compared with the nondiabetic control.(ABSTRACT TRUNCATED AT 250 WORDS)

L30 ANSWER 10 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 2

ACCESSION NUMBER: 2003:255166 BIOSIS
DOCUMENT NUMBER: PREV200300255166
TITLE: Fat modification in the diabetes diet.
AUTHOR(S): Julius, U. [Reprint Author]
CORPORATE SOURCE: Institut und Poliklinik fuer Klinische Stoffwechselforschung, Universitaetsklinikum, Fetscherstr. 74, 01307, Dresden, Germany
julius@rcs.urz.tu-dresden.de
SOURCE: Experimental and Clinical Endocrinology & Diabetes, (April 2003) Vol. 111, No. 2, pp. 60-65. print.
ISSN: 0947-7349.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 28 May 2003
Last Updated on STN: 28 May 2003

ED Entered STN: 28 May 2003
Last Updated on STN: 28 May 2003

AB The modification of dietary fat in the diet of diabetic patients is of interest with respect to metabolic and other consequences of this modification. To begin with the data are reviewed for the use of **monounsaturated fatty acids (MUFA)** in the diabetes diet. Compared to a carbohydrate-rich diet, glucose concentrations are lower. Blood pressure was also found to be lower. There were no major differences with respect to lipid concentrations. HDL-cholesterol levels tended to be higher after a **MUFA**-rich diet. In type-1 diabetic patients, the number of circulating big VLDL particles was greater after a **MUFA** diet than after a carbohydrate-rich diet. Comparisons were also made between diets enriched with **MUFA** and with polyunsaturated fatty acids (PUFA). With

respect to lipid concentrations, different groups observed different effects. While one group saw no differences in fasting lipids, they measured a higher remnant-like particle cholesterol after a diet enriched

-rich diet increased endothelium-dependent flow-mediated dilatation in the superficial femoral artery. **Alpha-linolenic acid** appears to be a precursor of eico-spentaenoic and **docosahexaenoic** fatty acids. As a diet rich in n-6 PUFA reduces this conversion, a n-6/n-3 PUFA ratio not exceeding 4-6 should be observed. No prospective data are available for **alpha-linolenic acid** in diabetic patients. The review summarizes the results of the Lyon Diet Heart Study and the Nurses' Health Study. Both studies saw a reduced cardiovascular risk associated with a higher intake of **alpha-linolenic acid**.
Finally, data on the effects of **fish oil** are given.
The latter has a clearly expressed **triglyceride**-lowering effect.
Data with respect to glucose control are heterogeneous. Major studies did not find any influence in glucose concentrations. Hepatic glucose production and peripheral insulin sensitivity remained constant.
Evidently, nerve function can be improved by **fish oil**.
Data have been compiled comparing the effects of **fish oil** with those of **olive oil**, **linseed oil** and **sunflower oil**.

L30 ANSWER 11 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:59067 BIOSIS
DOCUMENT NUMBER: PREV200400053144
TITLE: Acute effects of **monounsaturated fatty acids**, plant-derived n-3 or marine-derived n-3 polyunsaturated fatty acids on vascular endothelial function in **type-2 diabetes**.
AUTHOR(S): West, Sheila G. [Reprint Author]; Hecker, Kari D. [Reprint Author]; Mustad, Vikkie A.; Nicholson, Sue; Schoemer, Stephanie L. [Reprint Author]; Wagner, Paul [Reprint Author]; Hinderliter, Alan L.; Ulbrecht, Jan [Reprint Author]; Ruey, Peter; Kris-Etherton, Penny M. [Reprint Author]
CORPORATE SOURCE: Pennsylvania State Univ, University Park, PA, USA
SOURCE: Circulation, (October 28 2003) Vol. 108, No. 17 Supplement, pp. IV-782-IV-783. print.
Meeting Info.: American Heart Association Scientific Sessions 2003. Orlando, FL, USA. November 09-12, 2003. American Heart Association.
ISSN: 0009-7322 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Jan 2004
Last Updated on STN: 21 Jan 2004
ED Entered STN: 21 Jan 2004
Last Updated on STN: 21 Jan 2004

L30 ANSWER 12 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:270896 BIOSIS
DOCUMENT NUMBER: PREV200300270896
TITLE: **Fish oil**, diet regulation and regular exercise for management of hypertriglyceridemia.
AUTHOR(S): Al-Waili, Dr. Noori S. [Reprint Author]

CORPORATE SOURCE: Dubai Specialized Medical Center, Islamic Establishment for Education, P.O.Box 19099, Dubai, 04, United Arab Emirates

2003. FASEB.
ISSN: 0892-6638 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Jun 2003
Last Updated on STN: 11 Jun 2003
ED Entered STN: 11 Jun 2003
Last Updated on STN: 11 Jun 2003
AB 15 patients with high **triglyceride** (TG) , 9 females and 6 males (40-60 years) were entered for study. Six patients had adult **diabetes mellitus** and seven were overweight (index mass>28). Biochemical and hemtological tests were within normal range except for elevated TG in all patients and high blood sugar in six patients. The mean +/-SD fasting serum TG was 410 +/-78.8 mg/dl(range 300 to 550 mg/dl).The patients did not receive any medications for hypertriglyceridemia during two months preceding enrollment in the study. Each patient recieved program called Medicine of Balance which included diet regulation, regular exercise besides natural interventions (2 g of **fish oil** and 5 ml of **olive oil** a day). Linseed and **canola oils** were also used. The diet was composed of vegetables (60%), seeds (30%), fruits (10%) and fish (10%). 60 to 70% of food was uncooked. And the others were cooked by steamer. Exercise included walking for 30 min twice a day and running for 10 to 30 min every other day for those who could run. Mean serum TG was decreased to 232+/-53 mg/dl two months after starting program. 12 months later,serum TG ranged from 100 to 240 mg/dl (mean 168.3+/-45 mg/dl). Body weight decreased by 10 to 15% during first 6 months in all overweight patients.Other tests remained normal. Diabetic patients showed better sugar control. It might be concluded that diet regulation, **fish oil** and regular exercise are important as first step to treat hypertriglyceridemia.

L30 ANSWER 13 OF 40 HCAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 8
ACCESSION NUMBER: 1996:676741 HCAPLUS
DOCUMENT NUMBER: 125:327196
TITLE: Supplementation with **olive oil** rather than **fish oil** increases small arterial compliance in diabetic patients
AUTHOR(S): Bonnema, S. J.; Jespersen, L. T.; Marving, J.; Gregersen, G.
CORPORATE SOURCE: Department Internal Medicine, Esbjerg County Hospital, Esbjerg, DK-6700, Den.
SOURCE: Diabetes, Nutrition & Metabolism (1995), 8(2), 81-87
CODEN: DNMEEW; ISSN: 0394-3402
PUBLISHER: Editrice Kurtis
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 16 Nov 1996
AB The purpose of this study was to compare the effect of dietary supplementation with **fish oil** (polyunsatd. **omega-3 fatty acids**) or **olive oil** (**monounsatd. fatty acids**) on peripheral vascular compliance in diabetic human beings with incipient nephropathy. Twenty-eight patients with **diabetes mellitus** (15 men, 13 women, age: 43.8±14.1, diabetes duration: 17.8±11.8 (years±SD)) treated with insulin were randomized

double-blinded. They were to receive daily, over a 6 mo period, either 6 capsules of **fish oil**, or 6 capsules of **olive** oil, in addition to preexisting therapy. No patients were

HbA1c; and blood glucose. **Fish oil** increased fasting blood glucose compared to **olive oil** (baseline;end of trial: **fish oil**: 8.8±4.5; 9.2±4.1, **olive oil**: 10.7±4.5; 8.6±4.9 (mmol/l±SD), p<0.05). A decrease in arterial compliance was registered in the **fish oil** group; whereas an increase was registered in the **olive oil** group (baseline;end of trial: **fish oil**: 89±54; 68±35, **olive oil**: 84±69; 108±42 (µl/mmHg/100 mL±SD), p<0.01). No significant changes between the two groups were registered among the other parameters. It is concluded that **fish oil**, in contrast to **olive oil**, has an unfavorable effect on peripheral arterial compliance in patients with **diabetes mellitus** as well as deteriorating their blood glucose control.

L30 ANSWER 14 OF 40 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:886957 HCAPLUS
DOCUMENT NUMBER: 142:197114
TITLE: Plasma AA and **DHA** levels are not compromised in newly diagnosed gestational diabetic women
AUTHOR(S): Thomas, B.; Ghebremeskel, K.; Lowy, C.; Min, Y.; Crawford, M. A.
CORPORATE SOURCE: Institute of Brain Chemistry and Human Nutrition, London Metropolitan University, London, UK
SOURCE: European Journal of Clinical Nutrition (2004), 58(11), 1492-1497
CODEN: EJCNEQ; ISSN: 0954-3007
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 26 Oct 2004
AB OBJECTIVE: The polyunsatd. fatty acids, arachidonic (AA) and **docosahexaenoic** (**DHA**), are vital structural and functional components of the neural, vascular and visual systems. There is increased demand for these fatty acids during pregnancy. Diabetes impairs the synthesis of both AA and **DHA**. The authors have investigated the possibility that pregnancy-induced diabetes compromises the levels of plasma AA and **DHA** in newly diagnosed expectant mothers. DESIGN:: Cross-sectional study. SETTING: London, UK. SUBJECTS AND METHODS: Venous blood was obtained from 44 women with gestational **diabetes mellitus** (GDM) and from the same number of nondiabetics, during the third trimester. Fatty acid composition of plasma choline phosphoglycerides (CPG), **triglycerides** (TG) and cholesterol esters (CE) was analyzed. RESULTS: The GDM women had higher levels of AA (20:4n-6; P<0.0001) and AA/**linoleic acid** ratio (20:4n-6/18:2n-6; P<0.01) in the CPG, and **linoleic acid** (LA; P<0.0001), total n-6 (P<0.01), **DHA** (P<0.05) and n-3 metabolites (P<0.05) in TG compared to their nondiabetic counterparts. Similarly, AA (P<0.0001), osbond acid (22:5n-6; P<0.05), total n-6 metabolites (P<0.0001), AA/LA (P<0.0001) and n-6 metabolites/LA (P<0.01) were higher in the CE of the GDM women. There was no difference in the levels of **DHA** in CPG and CE between the 2 groups (P>0.05). CONCLUSIONS: The results of this study do not provide evidence that the activity of delta-6 or delta-5 desaturases, which are vital for the synthesis of AA and **DHA**, is compromised by pregnancy-induced diabetes. However, since the samples were taken at diagnosis, it is conceivable that the duration of the diabetes was too short to have a

discernable adverse effect on the levels of AA and **DHA** in plasma lipids.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS

DOCUMENT NUMBER: 141:123024
TITLE: Effects of a high-monounsaturated fat diet on glucose and lipid metabolisms in normal and diabetic mice
AUTHOR(S): Kotake, Jiro; Tanaka, Yoshiaki; Umehara, Norimitsu; Miyashita, Akira; Tsuru, Tomomitsu; Hikida, Shigeki; Mizote, Hiroyoshi
CORPORATE SOURCE: Central Research Laboratories, SSP Co., Ltd., Chiba, 286-8511, Japan
SOURCE: Journal of Nutritional Science and Vitaminology (2004), 50(2), 106-113
CODEN: JNSVA5; ISSN: 0301-4800
PUBLISHER: Center for Academic Publications Japan
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 14 May 2004
AB The beneficial effects of high-monounsatd. fat (high-**MUFA**) diets on diabetic patients have been reported, whereas studies concerning the effects on animals have been few. Although expts. on animals should be useful in elucidating underlying mechanisms, it is not clear even whether there are benefits of a high-**MUFA** diet in animals. This study examined the short-term effects of a high-**MUFA** diet on normal and genetically diabetic mice. The high-**MUFA** diet supplied 38% of the total calories as fat (26% from **MUFA**), while a regular diet was 13% fat (3% from **MUFA**). Normal C57BL/6J and diabetic C57BL/KsJ-db/db mice were fed either the regular or the high-**MUFA** diet for 1 wk. Serum glucose and lipid levels were then measured. In normal mice, hepatic **triglyceride** production was also compared between the 2 dietary groups using the Triton WR1339 method. An oral glucose tolerance test was conducted on the diabetic mice. After 1 wk of feeding to normal mice, the high-**MUFA** diet was seen to lower serum **triglyceride** levels and reduce hepatic **triglyceride** production in comparison with the regular diet; it is suggested that the lowering of **triglyceride** consists of mechanisms including reduced hepatic **triglyceride** production When diabetic mice were fed the high-**MUFA** diet with a controlled caloric intake, the serum glucose levels lowered without an accompanying deterioration in lipid metabolism and the **impaired glucose tolerance** was ameliorated. This study demonstrates that a high-**MUFA** diet can lower serum **triglyceride** levels in normal mice and improve disorders of glucose metabolism in diabetic mice.
REFERENCE COUNT: 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 16 OF 40 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2005:289372 HCAPLUS
DOCUMENT NUMBER: 142:291398
TITLE: Insulinomimetic fatty acids for antidiabetic application
INVENTOR(S): Carpinelli, Angelo Rafael; Curi, Rui; Roberta de Oliveira, Carla; Haber, Esther Piltcher
PATENT ASSIGNEE(S): Universidade de Sao Paulo, Brazil; Fundacao de Amparo a Pesquisa do Estado de Sao Paolo
SOURCE: Braz. Pedido PI, 39 pp.
CODEN: BPXXDX
DOCUMENT TYPE: Patent
LANGUAGE: Portuguese
FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO. KIND DATE APPLICATION NO. DATE

AB Fatty acids with up to 22 C atoms can have insulinomimetic or insulin-synergistic activities, activate protein phosphorylation and/or regulate protein levels and/or combat **hyperglycemia** and conditions associated with insulin deficiency or insulin resistance. Caprylic, palmitic, propionic, acetic, butyric, caproic, capric, lauric, myristic, stearic, oleic, linoleic, arachidonic, **eicosapentaenoic** , and **docosahexaenoic acids** are disclosed for antidiabetic use.

L30 ANSWER 17 OF 40 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:188812 HCAPLUS
DOCUMENT NUMBER: 139:358352
TITLE: Effects of cilostazol on serum lipid concentrations and plasma fatty acid composition in type 2 diabetic patients with peripheral vascular disease
AUTHOR(S): Nakamura, N.; Hamazaki, T.; Johkaji, H.; Minami, S.; Yamazaki, K.; Satoh, A.; Sawazaki, S.; Urakaze, M.; Kobayashi, M.; Osawa, H.; Yamabe, H.; Okomura, K.
CORPORATE SOURCE: The Second Department of Internal Medicine, Hirosaki University School of Medicine, 5 Zaifu-tyo, Hirosaki City, Aomori, 036-8562, Japan
SOURCE: Clinical and Experimental Medicine (2003), 2(4), 180-184
CODEN: CEMLBA; ISSN: 1591-8890
PUBLISHER: Springer-Verlag Italia Srl
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 11 Mar 2003
AB Cilostazol is an anti-thrombotic and vasodilating agent, reported to have both anti-thrombotic and cerebral vasodilating effects. We investigated the effects of cilostazol on serum lipid concns. and plasma fatty acid composition in type 2 diabetic patients with peripheral vascular disease. The serum concns. of total cholesterol, **triglycerides**, high-d. lipoprotein-cholesterol, lipoprotein (a), remnant-like particles-cholesterol, apolipoproteins, and plasma fatty acid composition were measured in 17 diabetic patients with peripheral vascular disease before and 1, 3, and 6 mo after administration of cilostazol (200 mg/day). Serum **triglyceride** concns. were significantly decreased after cilostazol (from 1.31±0.17 mmol/l to 0.86±0.07 mmol/l at 6 mo, $P<0.01$). Plasma **docosahexaenoic acid** levels were significantly increased after cilostazol (4.11±0.26% to 4.94±0.26% at 6 mo, $P<0.01$). Our findings show that cilostazol can induce some beneficial changes in serum lipid profile and plasma fatty acid composition
REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 18 OF 40 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:583496 HCAPLUS
DOCUMENT NUMBER: 137:350535
TITLE: Liver **triacylglycerols** and free fatty acids in streptozotocin-induced diabetic rats have atypical n-6 and n-3 pattern
AUTHOR(S): Ghebremeskel, K.; Bitsanis, D.; Koukkou, E.; Lowy, C.; Poston, L.; Crawford, M. A.
CORPORATE SOURCE: Institute of Brain Chemistry and Human Nutrition, University of North London, London, N7 8DB, UK
SOURCE: Comparative Biochemistry and Physiology, Part C:

Toxicology & Pharmacology (2002), 132C(3), 349-354
CODEN: CBPPFK; ISSN: 1532-0456
PUBLISHER: Elsevier Science Inc

docosahexaenoic (**DHA**) acids and a concomitant increase in linoleic (LA) and α-linolenic (ALA) acids. This metabolic perturbation is thought to be due to impaired activity of Δ6- and Δ5-desaturases. **Triacylglycerols** are the major lipid pool in plasma and liver tissue and have a significant influence on fatty acid composition of membrane and circulating phospholipids. Data on the distribution of n-6 and n-3 polyunsatd. fatty acids of **triacylglycerols** in diabetes are sparse. We investigated whether streptozotocin-induced diabetes in Sprague-Dawley rats alters fatty acid composition of **triacylglycerols** and free fatty acids of liver tissue. The animals were fed a breeding diet prior to mating, during pregnancy and lactation. On days 1-2 of pregnancy, diabetes was induced in 10 of the 25 rats. Liver was obtained at post partum day 16 for anal. Relative levels of LA (P=0.03), dihomo-gamma-**linolenic acid** (DHGLA) (P=0.02), AA (P=0.049), total n-6 (P=0.02), ALA (P=0.013), **eicosapentaenoic acid** (**EPA**) (P=0.004), docosapentaenoic acid (22:5n-3, DPA) (P=0.013), **DHA** (P=0.033), n-3 metabolites (P=0.015) and total n-3 (P=0.011) were significantly higher in the **triacylglycerols** of the diabetics compared with the controls. Similarly, liver free fatty acids of the diabetics had higher levels of LA (P=0.0001), DHGLA (P=0.001), AA (P=0.001), n-6 metabolites (P=0.002), total n-6 (P=0.0001), ALA (P=0.003), **EPA** (P=0.015), docosapentaenoic (22:5n-3, P=0.003), **DHA** (P=0.002), n-3 metabolites (P=0.005) and total n-3 (P=0.001). We conclude that impaired activity of desaturases and/or long chain acyl-CoA synthetase could not explain the higher levels of AA, **DHA** and n-6 and n-3 metabolites in the diabetics. This seems to be consistent with an alteration in the regulatory mechanism, which directs incorporation of polyunsatd. fatty acids either into **triacylglycerols** or phospholipids.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 19 OF 40 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1999:95070 HCAPLUS
DOCUMENT NUMBER: 130:294948
TITLE: Early lipid alterations in spontaneously diabetic rats
AUTHOR(S): de Gomez, Dumm I. N. T.; Montenegro, S.; Rarres, M. C.; Martinez, S. M.; Igal, R. A.
CORPORATE SOURCE: Instituto de Investigaciones Bioquimicas de La Plata, (INIBIOLP), Facultad de Ciencias Medicas, Universidad Nacional de La Plata, La Plata, 1900, Argent.
SOURCE: Acta Physiologica, Pharmacologica et Therapeutica Latinoamericana (1998), 48(4), 228-234
CODEN: APTLEZ; ISSN: 0327-6309
PUBLISHER: Acta Physiologica, Pharmacologica et Therapeutica Latinoamericana
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 12 Feb 1999
AB Human and exptl. **diabetes mellitus** extensively alters lipid metabolism The eSS is a rat strain that develops a spontaneous diabetes of slow evolution, resembling the non-insulin-dependent **diabetes mellitus** of young people. The authors report here disturbances in lipid metabolism of 5-mo old eSS rats compared to age-matched α-controls. Normal plasmatic glucose levels were found in the fasted state, whereas a diabetic curve was evident for eSS rats after

glucose load. **Triglyceride** content was elevated in plasma and in liver microsomal prepns. of eSS animals, when compared to the controls. The diabetic strain revealed a significant fall in the amount of

detected in phosphatidylcholine and phosphatidylethanolamine fractions from liver microsomes of eSS rats. The fatty acid pattern of eSS rat testis showed a raise in the relative percentage of arachidonic and a decrease in 22:5 (n-6), 22:5 (n-3) and 22:6 (n-3) acids compared to their controls. Diabetic rats exhibited a significant increase in microsomal cholesterol content and cholesterol/phospholipid ratio in liver and testis. In the latter tissue, higher values of fluorescence anisotropy were also observed The current observations indicate that in early stages of the diabetes onset, when eSS rats are still normoglycemic, severe alterations of lipid metabolism may contribute to the establishment and progression of the diabetic syndrome.

REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 20 OF 40 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1993:516224 HCAPLUS
DOCUMENT NUMBER: 119:116224
TITLE: Effect of **fish oil** concentrate on the lipoprotein profile in patients suffering from **diabetes mellitus** type II
AUTHOR(S): Herrmann, W.; Biermann, J.; Ratzmann, K. P.; Lindhofer, H. G.
CORPORATE SOURCE: Inst. Klin. Chem., Univ. Regensburg, Regensburg, D-W-8400, Germany
SOURCE: Horm. Lipoprotein Metab. (1993), 65-9. Editor(s): Steinmetz, Armin; Schneider, Juergen; Kaffarnik, Hans. Springer: Berlin, Germany.
CODEN: 59DZAE
DOCUMENT TYPE: Conference
LANGUAGE: English
ED Entered STN: 18 Sep 1993
AB In comparison to a **rapeseed oil** supplementation, a **fish oil** diet decreased the serum **triglyceride** concentration by 29%. LDL-cholesterol increased by 10%, HDL cholesterol by 9% (especially HDL2-cholesterol), and apo-B by 5%. Apo A-I was reduced by 9%. The fasting blood glucose and the glucose tolerance as well as the insulin levels (fasting and after load test) were unchanged. How far the pos. changes of the lipoprotein profile (a marked decrease in **triglycerides** and an increase of HDL-cholesterol) can be neutralized by a mild enhancement of LDL-cholesterol and apo-B has to be investigated by further studies.

L30 ANSWER 21 OF 40 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1993:494395 HCAPLUS
DOCUMENT NUMBER: 119:94395
TITLE: Effect of **fish oil** concentrate on the lipoprotein profile in patients suffering from **diabetes mellitus** type II
AUTHOR(S): Herrmann, W.; Biermann, J.; Ratzmann, K. P.; Lindhofer, H. G.
CORPORATE SOURCE: Bezirkskrankenhaus, Meiningen, O-6100, Germany
SOURCE: Mol. Biol. Atheroscler., [Ed. Proc. Eur. Atheroscler. Soc. Meet.] (1992), Meeting Date 1991, 249-52. Editor(s): Halpern, Manuel Judice. Libbey: London, UK.
CODEN: 58QDA6

DOCUMENT TYPE: Conference
LANGUAGE: English
ED Entered STN: 04 Sep 1992

was reduced by 9%. The fasting blood glucose and the glucose tolerance as the insulin level (fasting and after load test) were unchanged at the end of the verum period in comparison to the run-in-phase.

L30 ANSWER 22 OF 40 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1992:447251 HCAPLUS
DOCUMENT NUMBER: 117:47251
TITLE: Effect of **fish oil** concentrate on the lipoprotein profile in patients with **diabetes mellitus** type II
AUTHOR(S): Herrmann, Wolfgang; Biermann, Juergen; Ratzmann, Klaus Peter; Lindhofer, Hans Georg
CORPORATE SOURCE: Zentrallab., Bezirkskrankenhaus Meiningen, Meiningen, Germany
SOURCE: Medizinische Klinik (Muenchen, Germany) (1992), 87(1), 12-15
CODEN: MEKLA7; ISSN: 0723-5003
DOCUMENT TYPE: Journal
LANGUAGE: German
ED Entered STN: 08 Aug 1992
AB The influence of **fish oil** (FO) supplementation (6 g of capsules/day containing ≤3 g of **eicosapentaenoic + docosahexaenoic acids**) on blood serum glycerides and lipoproteins was monitored during a 12-wk. phase in non-insulin-dependent diabetics. Compared to controls receiving **rape oil**, the FO diet decreased **triglycerides** by 29%, and increased low- and high-d. lipoprotein cholesterol (I) by 9% resp., (especially HDL2-I) and apolipoprotein B (apo B) by 4%. Apolipoprotein A-I was reduced by 9%. Neither fasting glucose (II) nor the II-load test or insulin levels were affected. Thus, although the effects of FO on **triglycerides** and HDL-I were similar to those reported for normolipemic and hyperlipidemic subjects, the responses of LDL-I and apo B appear paradoxical. Possible mechanisms are discussed.

L30 ANSWER 23 OF 40 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1988:111110 HCAPLUS
DOCUMENT NUMBER: 108:111110
TITLE: Plasma **triacylglycerol** fatty acids in diabetic rats fed gamma-linolenic and marine n-3 fatty acids
AUTHOR(S): Huang, Yung Sheng; Horrobin, D. F.
CORPORATE SOURCE: Efamol Res. Inst., Kentville, NS, B4N 4H8, Can.
SOURCE: Medical Science Research (1987), 15(19), 1207-9
CODEN: MSCREJ; ISSN: 0269-8951
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 01 Apr 1988
AB Streptozotocin-diabetic rats were fed fat-free diets supplemented with 29% concentrate containing 84% γ- **linolenic acid** (18:3n-6) and 16% **linoleic acid** (18:2n-6), 2% **fish oil** concentrate containing 17.1% **eicosapentaenoic acid** (20:5n-3), 1.6% docosapentaenoic acid (22:5n-3) and 53.2% **docosahexaenoic acid** (22:6n-3), or 1% of each concentrate, and the fatty acid composition of plasma phospholipids, cholesterol esters, and **triglycerides** was compared with that of control rats fed the same diets and supplements. The lipid levels in diabetic and control rats on the same diet were similar, but phospholipid and **triglyceride**

levels were lower in both groups fed the n-3 fatty acids. Diabetes-induced changes in saturated and **monounsatd. fatty** acids of plasma lipids were not affected by diet. In diabetic

n-6. In phospholipids, arachidonic acid (20:4n-6) levels were unchanged and 18:2n-6, 18:3n-6, and eicosatrienoic acid (20:3n-6) were increased. This suggests that Δ6-desaturase and Δ5-desaturase are inhibited in diabetes. Diabetes accentuated the suppression of Δ5-desaturase activity found with the n-3 fatty acid diet. In cholesterol esters in diabetes, n-3 fatty acids were lower than in phospholipids.

L30 ANSWER 24 OF 40 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1987:403539 HCAPLUS
DOCUMENT NUMBER: 107:3539
TITLE: High-performance liquid chromatographic analysis of serum long-chain fatty acids by direct derivatization method
AUTHOR(S): Miwa, Hiroshi; Yamamoto, Magobei; Nishida, Tatsuro; Nunoi, Kiyohide; Kikuchi, Masanori
CORPORATE SOURCE: Fac. Pharm. Sci., Fukuoka Univ., Fukuoka, 814-01, Japan
SOURCE: Journal of Chromatography (1987), 416(2), 237-45
CODEN: JOCRAM; ISSN: 0021-9673
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 11 Jul 1987
AB A new visible-UV labeling method for the HPLC anal. in serum of individual free fatty acids, including polyunsatd. fatty acids, is described. Without commonly used isolation steps, fatty acids in serum were directly derivatized by treatment with acidic 2-nitrophenylhydrazine HCl. The derivatized fatty acids were extracted into n-hexane and separated isocratically on a reversed-phase C8 column within 15 min. The detection limits ranged from 400 fmol to 1 pmol and from 100 to 200 fmol per injection with visible and UV detection, resp. Visible detection had better selectivity, and free fatty acid levels were determined in sera obtained from healthy controls and patients with **diabetes mellitus**. In all the subjects studied, the precise quantitation could be performed with 25 μL serum. Anal. recoveries ranged 98.3-103.4%. The intra- and interassay relative standard deviations were <2.7 and 3.5%, resp. The present method is superior to the previously published methods for routine analyses: it is cheaper, the procedure is simpler, the anal. time is shorter and both resolution and sensitivity are better.

L30 ANSWER 25 OF 40 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1986:459854 HCAPLUS
DOCUMENT NUMBER: 105:59854
TITLE: Correlations between unsaturated fatty acids and medium C-chain **triglycerides**
AUTHOR(S): Caponnetto, A.; Pagano, M. A.; Rondinone, R.; Zunin, P.
CORPORATE SOURCE: Fac. Med., Univ. Genova, Genoa, Italy
SOURCE: Bollettino - Societa Italiana di Biologia Sperimentale (1986), 62(2), 249-56
CODEN: BSIBAC; ISSN: 0037-8771
DOCUMENT TYPE: Journal
LANGUAGE: Italian
ED Entered STN: 23 Aug 1986
AB Rats were fed diets high in unsatd. fatty acids and/or in **MCT** (**medium-chain triglycerides**) and subsequently

subjected to subtotal hepatectomy or injected with alloxan to induce diabetes, and fatty acid composition of dietary **oleic acid** [112-80-1] reduced linoleic acid [60-33-3] in the

Δ9-desaturase. It also reduced ω3- **docosahexaenoic acid** [6217-54-5] in the liver only of the treated alloxan rats; it may affect desaturase.

L30 ANSWER 26 OF 40 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1987:29472 HCAPLUS
DOCUMENT NUMBER: 106:29472
TITLE: Quantitative determination of the fatty acid composition of human serum lipids by high-performance liquid chromatography
AUTHOR(S): Shimomura, Yoshiharu; Sugiyama, Satoru; Takamura, Tadanobu; Kondo, Taizo; Ozawa, Takayuki
CORPORATE SOURCE: Fac. Med., Univ. Nagoya, Nagoya, 466, Japan
SOURCE: Journal of Chromatography (1986), 383(1), 9-17
CODEN: JOCRAM; ISSN: 0021-9673
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 07 Feb 1987
AB An HPLC method for the anal. of the fatty acid composition of human serum lipids with fluorescence detection was examined Both free and total fatty acids extracted from serum were derivatized with 9-anthryldiazomethane and were analyzed using MeOH-H2O (94.7:5.3) as mobile phase. Twelve kinds of fatty acid were detected, both in the free and total fatty acids, and were well separated Concns. of individual fatty acids of serum lipids were estimated from an internal standard, heptadecanoic acid. The results correlated well with those from 2 other quant. analyses. HPLC anal. of fatty acids is a reliable method for determining individual fatty acids of human serum lipids. The compns. of free fatty acids and total fatty acids of serum lipids were analyzed and compared in normal subjects, diabetics, and angina pectoris patients by this method.

L30 ANSWER 27 OF 40 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1979:52659 HCAPLUS
DOCUMENT NUMBER: 90:52659
TITLE: **Triglyceride** fatty acid patterns in severe diabetic macroangiopathy
AUTHOR(S): Singer, P.; Gnauck, G.; Honigmann, G.; Schliack, V.
CORPORATE SOURCE: Zentralinst. Herz- Kreislauf-Regulationsforsch., DAW, Berlin, Ger. Dem. Rep.
SOURCE: Deutsche Gesundheitswesen (1978), 33(46), 2179-83
CODEN: DEGEA3; ISSN: 0012-0219
DOCUMENT TYPE: Journal
LANGUAGE: German
ED Entered STN: 12 May 1984
AB The percentage of palmitic and **linoleic acids** was higher and that of palmitoleic and **oleic acids** lower in the serum than in arterial and adipose tissues of 16 diabetic patients on insulin therapy after leg amputation secondary to severe macroangiopathy and gangrene. The serum and arterial wall content of eicosatrienoic, arachidonic, and **eicosapentaenoic acids** (precursors of prostaglandins) was the same but was higher than in adipose tissue. In view of the differences in the fatty acid pattern between arterial wall on the one hand and serum and adipose tissue on the other hand, no conclusions on the arterial vessel pattern can be extrapolated from serum levels. The results are discussed in relation to arteriosclerosis.

L30 ANSWER 28 OF 40 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

SOURCE: Pharmaceutical Journal, (20 Nov 2004) Vol. 273, No. 7326, pp. 750-752.
Refs: 17
ISSN: 0031-6873 CODEN: PHJOAV
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Note
FILE SEGMENT: 003 Endocrinology
008 Neurology and Neurosurgery
017 Public Health, Social Medicine and Epidemiology
018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20041228
Last Updated on STN: 20041228
ED Entered STN: 20041228
Last Updated on STN: 20041228
AB It is important to ensure that any fat consumed is of a beneficial type. More emphasis should be placed on **MUFAs** and n3 PUFAs to replace both SFAs and n6 PUFAs. This will help to ensure an appropriate balance of n3 to n6 PUFAs and a reduced intake of SFAs. This could help to reduce the risk of CVD and other chronic conditions with an inflammatory component. Irrespective of the type of fatty acids contained, all fats provide 9kcal (37kJ) per gram, making fat the most concentrated source of energy in the diet and a potentially significant risk factor for obesity so, although some fatty acids are essential for health, total fat intake should still be limited. Topping up on EFAs is best done through dietary measures. Usually, it is the n3 EFAs that are needed. Hence the best advice is to eat n3-rich oily fish and seeds or seed oils (see Panel 1, p749). For people who do not like eating oily fish and who are concerned about the risk of CVD, a supplement providing 1g of n3 fatty acids (from **fish oils**) can be suggested. If supplements are used, because of their instability, it is best to buy them in small quantities and to keep them refrigerated.

L30 ANSWER 29 OF 40 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2004386061 EMBASE
TITLE: n-3 long chain polyunsaturated fatty acids: A nutritional tool to prevent insulin resistance associated to **type 2 diabetes** and obesity?.
AUTHOR: Delarue J.; LeFoll C.; Corporeau C.; Lucas D.
CORPORATE SOURCE: J. Delarue, EA-948 Oxylipides, Faculte de Medecine, 29200 Brest, France. jacques.delarue@univ-brest.fr
SOURCE: Reproduction Nutrition Development, (2004) Vol. 44, No. 3, pp. 289-299.
Refs: 64
ISSN: 0926-5287 CODEN: RNDEE5
COUNTRY: France
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 003 Endocrinology
005 General Pathology and Pathological Anatomy
006 Internal Medicine
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English

SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20040930
Last Updated on STN: 20040930

docosahexaenoic acid (**DHA**, 22:6 n-3), are present in mammal tissues both from endogenous synthesis from desaturation and elongation of 18:3 n-3 and/or from dietary origin (marine products and **fish oils**). In rodents in vivo, n-3 LC-PUFA have a protective effect against high fat diet induced insulin resistance. Such an effect is explained at the molecular level by the prevention of many alterations of insulin signaling induced by a high fat diet. Indeed, the protective effect of n-3 LC-PUFA results from the following: (a) the prevention of the decrease of phosphatidyl inositol 3' kinase (PI3 kinase) activity and of the depletion of the glucose transporter protein GLUT4 in the muscle; (b) the prevention of the decreased expression of GLUT4 in adipose tissue. In addition, n-3 LC-PUFA inhibit both the activity and expression of liver glucose-6-phosphatase which could explain the protective effect with respect to the excessive hepatic glucose output induced by a high fat diet. n-3 LC-PUFA also decrease muscle intramyofibrillar **triglycerides** and liver steatosis. This last effect results on the one hand, from a decreased expression of lipogenesis enzymes and of delta 9 desaturase (via a depleting effect on sterol response element binding protein 1c (SREBP-1c). On the other hand, n-3 LC-PUFA stimulate fatty acid oxidation in the liver (via the activation of peroxisome proliferator activated receptor α (PPAR-α)). In patients with **type 2 diabetes**, **fish oil** dietary supplementation fails to reverse insulin resistance for unclear reasons, but systematically decreases plasma **triglycerides**. Conversely, in healthy humans, **fish oil** has many physiological effects. Indeed, **fish oil** reduces insulin response to oral glucose without altering the glycaemic response, abolishes extraggression at times of mental stress, decreases the activation of sympathetic activity during mental stress and also decreases plasma **triglycerides**. These effects are encouraging in the perspective of prevention of insulin resistance but further clinical and basic studies must be designed to confirm and complete our knowledge in this field.

L30 ANSWER 30 OF 40 EMBASE
on STN
ACCESSION NUMBER: 2004299125 EMBASE
TITLE: Nutrition therapy for dyslipidemia.
AUTHOR: Carson J.A.S.
CORPORATE SOURCE: Dr. J.A.S. Carson, Department of Clinical Nutrition, Center for Human Nutrition, Univ. of Texas SW Medical Center, 5323 Harry Hines Boulevard, Dallas, TX 75390-8877, United States. joann.carson@utsouthwestern.edu
SOURCE: Current Diabetes Reports, (2003) Vol. 3, No. 5, pp. 397-403.
Refs: 50
ISSN: 1534-4827
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 003 Endocrinology
006 Internal Medicine
017 Public Health, Social Medicine and Epidemiology
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 20040729
Last Updated on STN: 20040729
ED Entered STN: 20040729

should also be limited. Incorporation of functional foods, such as stanol-containing margarine, soy products, and soluble fiber-rich cereals and vegetables can provide further benefit. In addition to weight loss and physical activity, individuals with hypertriglyceridemia benefit from a diet moderate in fat and carbohydrate rather than a low-fat diet. Including monounsaturated or **omega-3 fatty acids** lowers serum **triglycerides**. Many of the dietary strategies to optimize serum lipids also contribute to glycemic control in patients with **diabetes mellitus**.

L30 ANSWER 31 OF 40 EMBASE
on STN
ACCESSION NUMBER: 2003492682 EMBASE
TITLE: n-3 fatty acids and the risk of sudden cardiac death: Emphasis on heart rate variability.
AUTHOR: Christensen J.H.
CORPORATE SOURCE: J.H. Christensen, Department of Nephrology, Aalborg Hospital, DK-9100 Aalborg, Denmark
SOURCE: Danish Medical Bulletin, (2003) Vol. 50, No. 4, pp. 347-367.
Refs: 373
ISSN: 0907-8916 CODEN: DMBUAE
COUNTRY: Denmark
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 017 Public Health, Social Medicine and Epidemiology
018 Cardiovascular Diseases and Cardiovascular Surgery
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20040116
Last Updated on STN: 20040116
ED Entered STN: 20040116
Last Updated on STN: 20040116
AB Marine n-3 PUFA have potential antiatherogenic, antithrombotic, and antiinflammatory properties. However, recent research have addressed the antiarrhythmic effect of these fatty acids as a major explanation for their beneficial role in IHD and such an effect may explain the reduction in the incidence of SCD observed among fish eaters. SCD remains a serious problem in the Western Countries and although prevention of SCD is one of the main targets in modern cardiology the incidence of SCD has not declined during the past decades. Therefore, there is a need for other approaches to reduce the incidence of SCD. Thus, the aims of this study were 1) to study the impact of n-3 PUFA on 24-hour HRV, a recognized predictor of arrhythmic events and SCD in high-risk patients and in healthy subjects, and 2) to review the current knowledge about n-3 PUFA and the risk of SCD in humans and the proposed actions of n-3 PUFA responsible for an antiarhythmic effect. Subjects eating a modest amount of marine n-3 PUFA have an approximately 50% reduction in the risk of SCD compared to subjects not eating fish and in one study there was a close negative association between the risk of SCD and the cellular level of n-3 PUFA. Two large intervention studies support a beneficial effect of n-3 PUFA on the risk of SCD. In the DART study from 1989 a significant 29% reduction was found among post-MI men advised to eat fatty fish twice a week compared to those not advised so. This reduction could not be explained by an antiatherosclerotic or an antithrombotic effect of n-3

PUFA and an antiarrhythmic effect was considered operative. Ten years later the GISSI Prevenzione trial showed a 45% reduction in SCD among post-MI patients given one fish oil capsule daily.

PUFA on SCD in humans it is of importance to investigate if n-3 PUFA have actions in humans comparable to data from non-human studies. A surrogate for the risk of developing ventricular arrhythmias and SCD in humans is 24-hour HRV. Thus, in patients with IHD the risk of malignant ventricular arrhythmias and SCD is increased with decreased HRV. On the opposite, pharmacological interventions resulting in an improved patient survival have been associated with an increased HRV. In our studies we found positive associations between cellular levels of marine n-3 PUFA and HRV in post-MI patients and in patients referred for coronary angiography suspected of IHD. Also, in these patients cellular levels of marine n-3 PUFA were independently correlated with HRV. When post-MI patients were given 5.2 g of marine n-3 PUFA daily for 12 weeks their HRV significantly increased. These findings may help explain why marine n-3 PUFA offer protection against SCD in patients with IHD. Patients with CRF and patients with DM comprise patient populations with an increased risk of SCD and an attenuated HRV. In these two groups of patients we found a close positive association between the cellular level of marine n-3 PUFA and HRV suggesting a beneficial effect of marine n-3 PUFA on HRV. Further research with dietary intervention with n-3 PUFA to CRF and DM patients should clarify if this effect can be translated into a reduction of coronary events. A decreased HRV may predict a poor outcome among healthy subjects due to an increased risk of SCD. We found a close positive association between cellular levels of marine n-3 PUFA and HRV in healthy men but not in healthy women. Dietary intervention with either 2.0 g or 6.6 g of marine n-3 PUFA daily for 12 weeks revealed a dose-dependent increase in HRV among men with a low base-line HRV. The results may help explain why dietary marine n-3 PUFA may reduce the risk of SCD in healthy men. It is a novel observation that n-3 PUFA have a beneficial impact on HRV in humans. The results from non-human studies showing effects of n-3 PUFA on sodium channels, calcium-channels and adrenergic receptors may, if applicable to humans, explain this effect of n-3 PUFA on HRV. However, n-3 PUFA may also cause a central modulation of HRV and, n-3 PUFA may thus modulate HRV both at the level of the brain and in the heart. In conclusion, the data suggest that marine n-3 PUFA have a beneficial impact on HRV in patients at high risk of SCD and in healthy men. Furthermore, our data may indicate that the protective effect of n-3 PUFA on SCD found among post-MI patients and healthy subjects is caused by a modulation of autonomic control with increased vagal tone. Therefore, given the safety and low cost of implementing a modest amount of marine n-3 PUFA in the diet, an adequate dietary fish intake may have a significant role to play in the primary and secondary prevention of out-of-hospital SCD.

L30 ANSWER 32 OF 40 EMBASE
on STN

ACCESSION NUMBER: 2003487298 EMBASE
TITLE: Prudent Diet and Preventive Nutrition from Pediatrics to Geriatrics: Current Knowledge and Practical Recommendations.
AUTHOR: Enas E.A.; Senthilkumar A.; Chennikkara H.; Bjurlin M.A.
CORPORATE SOURCE: Dr. E.A. Enas, CADI Research Foundation, 1935, Green Trails, Lisle, IL 60523, United States. cadiusa@msn.com
SOURCE: Indian Heart Journal, (2003) Vol. 55, No. 4, pp. 310-338. Refs: 394
ISSN: 0019-4832 CODEN: IHEJAG
COUNTRY: India
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery

029 Clinical Biochemistry
LANGUAGE: English
ENTRY DATE: Entered STN: 20040105

L30 ANSWER 33 OF 40 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2002387355 EMBASE
TITLE: Effects of purified **eicosapentaenoic** and **docosahexaenoic acids** on glycemic control, blood pressure, and serum lipids in type 2 diabetic patients with treated hypertension.
AUTHOR: Woodman R.J.; Mori T.A.; Burke V.; Puddey I.B.; Watts G.F.; Beilin L.J.
CORPORATE SOURCE: R.J. Woodman, Department of Medicine, University of Western Australia, PO Box X2213, Perth, WA 6847, Australia. rwoodman@cyllene.uwa.edu.au
SOURCE: American Journal of Clinical Nutrition, (1 Nov 2002) Vol. 76, No. 5, pp. 1007-1015.
Refs: 61
ISSN: 0002-9165 CODEN: AJCNAC
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20021121
Last Updated on STN: 20021121
ED Entered STN: 20021121
Last Updated on STN: 20021121
AB Background: n-3 Fatty acids lower blood pressure, improve lipids, and benefit other cardiovascular disease risk factors. Effects on glycemia in patients with **type 2 diabetes** are uncertain.
Objective: We determined whether purified **eicosapentaenoic acid** (**EPA**) and **docosahexaenoic acid** (**DHA**) have differential effects on glycemic control, including insulin sensitivity and stimulated insulin secretion; 24-h ambulatory blood pressure; and serum lipids in type 2 diabetic patients with treated hypertension. Design: In a double-blind, placebo-controlled trial of parallel design, 59 subjects were randomly assigned to consume 4 g **EPA**, **DHA**, or **olive oil**/d for 6 wk while continuing to consume their usual diet. Results: Thirty-nine men and 12 postmenopausal women with a mean (±SE) age of 61.2 ± 1.2 y completed the study. In comparison with the change from baseline in fasting glucose in the **olive oil** group, fasting glucose in the **EPA** and **DHA** groups increased 1.40 ± 0.29 mmol/L (P = 0.002) and 0.98 ± 0.29 mmol/L (P = 0.002), respectively. Neither **EPA** nor **DHA** had significant effects on glycated hemoglobin, fasting insulin or C-peptide, insulin sensitivity or secretion, or blood pressure. Serum **triacylglycerols** in the **EPA** and **DHA** groups decreased 19% (P = 0.022) and 15% (P = 0.022), respectively. There were no significant changes in serum total, LDL, or HDL cholesterol, although HDL(2) cholesterol in the **EPA** and **DHA** groups increased 16% (P = 0.026) and 12% (P = 0.05), respectively. HDL(3) cholesterol decreased 11% (P = 0.026) with **EPA** supplementation.
Conclusions: **EPA** and **DHA** had similar benefits on lipids but adverse effects on short-term glycemic control in hypertensive

diabetic patients. The overall implications for cardiovascular disease require long-term evaluation.

TITLE: Dietary fatty acids in the management of **diabetes mellitus**.
AUTHOR: Berry E.M.
CORPORATE SOURCE: E.M. Berry, Dept. of Human Nutri. and Metabolism, Hebrew University-Hadassah Med. Sch., POB 12272, Jerusalem 91120, Israel
SOURCE: American Journal of Clinical Nutrition, (1997) Vol. 66, No. 4 SUPPL., pp. 991S-997S.
Refs: 64
ISSN: 0002-9165 CODEN: AJCNAC
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 003 Endocrinology
006 Internal Medicine
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 971113
Last Updated on STN: 971113
ED Entered STN: 971113
Last Updated on STN: 971113
AB Dietary fatty acid recommendations for patients with **diabetes mellitus** may be neither similar to, nor extrapolated from, those for the normal population: some evidence suggests that diabetes prevalence may be correlated with the dietary ratio of n-6 to n-3 fatty acids. In human experiments, n-3 fatty acids may improve many of the metabolic sequelae of insulin resistance by lowering blood pressure and **triacylglycerol** concentrations. In animals, n- 3 fatty acids may causeless weight gain than other fats; however, they may raise low-density-lipoprotein concentrations, increase hepatic glucose output, and decrease insulin secretion in non-insulin-dependent **diabetes mellitus**. In a minority of patients with insulin-dependent **diabetes mellitus**, glycemic control may be adversely affected. n-6 Fatty acids lower plasma cholesterol but may increase lipoprotein oxidation. Glucose in the presence of transition metals may produce free radicals and result in pancreatic damage and the formation of glycosylation products that inhibit nitric oxide- mediated smooth muscle relaxation; **fish oil** may counter these effects. High-carbohydrate, low-fat diets, once recommended for **diabetes mellitus**, appear to aggravate hypertriglyceridemia and are inferior to diets high in **monounsaturated fatty acids** (**MUFAs**) if saturated fats are kept to a minimum. **MUFA**-rich diets improve lipid profiles and may also have antioxidant properties. However, high-fat diets-whatever their composition-promote obesity. Current advice individualizes carbohydrate and fat requirements to optimize blood glucose and lipid concentrations in a lifestyle program in control obesity, exercise, smoking, and blood pressure. Fatty acid modifications may finetune the diet if proper balance is kept between the different long-chain polyunsaturated fatty acids and antioxidant requirements.

L30 ANSWER 35 OF 40 EMBASE
on STN
ACCESSION NUMBER: 95019146 EMBASE
DOCUMENT NUMBER: 1995019146
TITLE: Dietary supplementation with n-3 fatty acids increases gluconeogenesis from glycerol but not hepatic glucose

production in patients with non- insulin-dependent **diabetes mellitus**.

AUTHOR: Puhakainen I.; Ahola T.; Yki-Jarvinen H.

ISSN: 0002-9165 CODEN: AJCNAC
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
029 Clinical Biochemistry
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 950201
Last Updated on STN: 950201
ED Entered STN: 950201
Last Updated on STN: 950201
AB **Fish-oil** supplementation decreases serum **triacylglycerols** but may worsen **hyperglycemia** in patients with non-insulin-dependent **diabetes mellitus**. The reason for the possible deterioration of glycemia is unclear. We examined whether inhibition of **triacylglycerol** synthesis by n-3 fatty acids changes lipolysis, glycerol gluconeogenesis, or fatty acid oxidation. Nine obese patients with non-insulin-dependent **diabetes mellitus** participated in a randomized double-blind crossover study in which 6 wk of n-3 fatty acid supplementation (12 g **fish oil**) was compared with 6 wk of corn plus **olive oil**. Serum **triacylglycerols** decreased by 30% during n-3 fatty acid supplementation. Glycerol gluconeogenesis ([U-14C]glycerol) increased by 32%. However, overall glucose production ([3-3H]glucose), glycemic control, and fatty acid oxidation remained unchanged. Thus, 6 wk of n-3 fatty acid supplementation lowers **triacylglycerols** in patients with non-insulin- dependent **diabetes mellitus** without worsening glycemic control. However, n-3 fatty acid supplementation increases glycerol gluconeogenesis, which could contribute to deterioration of glycemic control during long-term treatment with high doses of **fish-oil** supplements.

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ACCESSION NUMBER: 93236711 EMBASE
DOCUMENT NUMBER: 1993236711
TITLE: Abnormal serum fatty acid composition in non-insulin-dependent **diabetes mellitus**
AUTHOR: Bohov P.; Gelienova K.; Sebokova E.; Klimes I.
CORPORATE SOURCE: Inst of Experimental Pharmacology, Slovak Academy of Sciences, Dubravska Str 9,842 16 Bratislava, Slovakia
SOURCE: Annals of the New York Academy of Sciences, (1993) Vol. 683, pp. 367-370.
ISSN: 0077-8923 CODEN: ANYAA
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 003 Endocrinology
029 Clinical Biochemistry
LANGUAGE: English
ENTRY DATE: Entered STN: 930912
Last Updated on STN: 930912
ED Entered STN: 930912
Last Updated on STN: 930912
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L30 ANSWER 37 OF 40 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

AUTHOR: Storlien L.H.; Pan D.A.; Kriketos A.D.; Baur L.A.
CORPORATE SOURCE: Dept of Medicine (Endocrinology), University of Sydney,Sydney, NSW 2006, Australia
SOURCE: Annals of the New York Academy of Sciences, (1993) Vol. 683, pp. 82-90.
ISSN: 0077-8923 CODEN: ANYAA
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 003 Endocrinology
029 Clinical Biochemistry
LANGUAGE: English
ENTRY DATE: Entered STN: 930912
Last Updated on STN: 930912
ED Entered STN: 930912
Last Updated on STN: 930912
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L30 ANSWER 38 OF 40 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
ACCESSION NUMBER: 75135940 EMBASE
DOCUMENT NUMBER: 1975135940
TITLE: The fatty acid pattern of adipose tissue and liver **triglycerides** according to fat droplet size in liver parenchymal cells of diabetic subjects.
AUTHOR: Singer P.; Gnauck G.; Honigmann G.; et al.
CORPORATE SOURCE: Clin. Diab. Metab. Dis., Cent. Diab. Metab. Dis., Humboldt Univ., Berlin, Germany
SOURCE: Diabetologia, (1974) Vol. 10, No. 5, pp. 455-458.
CODEN: DBTGAJ
DOCUMENT TYPE: Journal
FILE SEGMENT: 003 Endocrinology
006 Internal Medicine
LANGUAGE: English
AB In 40 diabetic inpatients the fatty acid pattern of **triglycerides** in liver fat and in subcutaneous adipose tissue was determined by gas liquid chromatography. With rising size of fat droplets in liver parenchymal cells there was a significant increase of palmitic acid (C:16), **oleic acid** (C 18:1) and **linoleic acid** (C 18:2) as well as a decrease of myristoleic acid (C 14:1), arachidonic acid (C 20:4) and **eicosapentaenoic acid** (C 20:5) in liver **triglycerides**, resulting in a fatty acid composition in big droplets similar to that of adipose tissue. Fatty acid pattern of subcutaneous depot fat was strikingly constant. (39 references).

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on STN
ACCESSION NUMBER: 75079447 EMBASE
DOCUMENT NUMBER: 1975079447
TITLE: The fatty acid composition of the **triglycerides** in liver and subcutaneous fat in diabetics in dependence on liver fatty degeneration.
AUTHOR: Singer P.; Gnauck G.; Honigmann G.
CORPORATE SOURCE: Clin. Diab. Metab. Dis., Kaulsdorf Hosp., Berlin, Germany
SOURCE: Acta Medica Polona, (1974) Vol. 15, No. 1-2, pp. 27-37.
CODEN: AMDPAA
DOCUMENT TYPE: Journal

FILE SEGMENT: 006 Internal Medicine
005 General Pathology and Pathological Anatomy
029 Clinical Biochemistry

whereas 44 diabetics revealed fatty degeneration of the liver parenchyma cells of various stages. In normal liver medium chain fatty acids, myristoleic acid and long chain fatty acids (C 20 and above), especially **eicosapentaenoic acid** (C 20:5), were elevated and palmitic, oleic, linoleic and **linolenic acid** were decreased significantly in comparison to the subcutaneous adipose tissue. In fatty degeneration of the liver parenchyma palmitic, oleic and **linoleic acid** increased, on the other hand arachidonic and **eicosapentaenoic acid** diminished significantly, the size of the fat droplets in the liver parenchyma cells being the best reference for correlation. With rising size of the droplets in the liver the **triglyceride** fatty acid pattern gets similar to that of the adipose tissue. The results are briefly discussed.

L30 ANSWER 40 OF 40 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
ACCESSION NUMBER: 2005-195966 [20] WPIDS
DOC. NO. CPI: C2005-062090
TITLE: Oral solid dosage form used for treating osteoporosis and **diabetes mellitus** comprises fatty acid salt particles having specific diameter and biologically active agent.
DERWENT CLASS: B05 B07
INVENTOR(S): OPAWALE, F; SOLTERO, R
PATENT ASSIGNEE(S): (NOBE-N) NOBEX CORP
COUNTRY COUNT: 108
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2005016312	A1	20050224	(200520)*	EN	45

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2005016312	A1	WO 2004-US26403	20040813

PRIORITY APPLN. INFO: US 2003-494821P 20030813
ED 20050324
AN 2005-195966 [20] WPIDS
AB WO2005016312 A UPAB: 20050324
NOVELTY - Oral solid dosage form (C1) comprises fatty acid salt particles having a size distribution, where the particles are 1-1000 micro m in diameter.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for an oral composition comprising (C1) and a biologically active agent.
ACTIVITY - Osteopathic; Antidiabetic. No biological data given.
MECHANISM OF ACTION - None given.
USE - Used for treating osteoporosis and **diabetes**

mellitus (claimed).
ADVANTAGE - The oral solid dosage form allows effective delivery of polypeptide pharmaceuticals that are prone to enzymatic [illegible]

=> file stnguide
COST IN U.S. DOLLARS — SINCE FILE ENTRY: 94.53 — TOTAL SESSION: 268.50

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) — SINCE FILE ENTRY: -10.95 — TOTAL SESSION: -15.33

FILE 'STNGUIDE' ENTERED AT 11:23:32 ON 22 APR 2005
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Apr 15, 2005 (20050415/UP).

=> d cost
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
CONNECT CHARGES	0.00	140.33
NETWORK CHARGES	0.78	4.80
DISPLAY CHARGES	0.00	124.15
FULL ESTIMATED COST	0.78	269.28

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) — SINCE FILE ENTRY: 0.00 — TOTAL SESSION: -15.33

IN FILE 'STNGUIDE' AT 11:31:15 ON 22 APR 2005

=>
=> d his

(FILE 'HOME' ENTERED AT 10:45:15 ON 22 APR 2005)

FILE 'HCAPLUS' ENTERED AT 10:45:27 ON 22 APR 2005
E HEIRLER H/AU
L1 2 S E4

FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS' ENTERED AT 10:46:14 ON 22 APR 2005
L2 624438 S (DIABETES(W)MELLITUS) OR "TYPE 1 DIABETES" OR "TYPE 2 DIABETE
L3 316701 S (MEDIUM(W)CHAIN(W)TRIGLYCERIDE?) OR TRIACYLGLYCEROL? OR TRIGL
L4 198146 S (OLEIC ACID?) OR "9-OCTADECENOIC ACID" OR OLEATE? OR (OLIVE O
L5 122681 S (LINOLEIC ACID?) OR LINOLEATE? OR "9,12-OCTADECADIENOIC ACID"
L6 2949 S (Ω)(W)"6"(W)FATTY(W)ACID? OR (DOUBLE(W)UNSATURATED(W)TR
L7 85651 S ((A)(W)LINOLENIC(W)ACID?) OR (LINOLENIC ACID?) OR LINOL
L8 1 S (TRIPLE(W)UNSATURATED(W)TRIGLYCERIDE?)
L9 52757 S EICOSAPENTAEN? OR EICOSAPENTAENOIC ACID? OR TIMNODONIC ACID?
L10 60428 S DOCOSAHEXAEN? OR DOCOSAHEXAENOIC ACID? OR "DHA" OR (FISH OIL?
L11 4405 S (SATURATED(W)LONG(W)CHAIN(W)(TRIGLYCERIDE? OR TRIACYLGLYCEROL
L12 152425 S EMULSIFIER OR EMULSIF?
L13 69467 S MONOGLYCERIDE? OR MONOACYLGLYCEROL? OR DIGLYCERIDE? OR DIACYL
L14 421736 S "VITAMIN A" OR RETINOL? OR RETINYL PALMITATE? OR "VITAMIN D"

```
L15         48454 S CAROTIN? OR (BETA(W)(CAROTIN? OR CAROTENE?)) OR ((B)(W)
L16         27388 S FLAVORING? OR (BUTTER(W)FLAVOR?) OR ROSEMARY? OR (ROSEMARY(W)
L17        130972 S RETINYL PALMITATE? OR "VITAMIN D3" OR CHOLECALCIFEROL? OR "VI
```

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L22           699 S L21 AND L4
L23           204 S L22 AND (L5 OR L6 OR L7 OR L8)
L24            73 S L23 AND (L9 OR L10)
L25             1 S L24 AND L11
L26            14 S L24 AND (L12 OR L13 OR L14 OR L15 OR L16)
L27            11 DUP REM L26 (3 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 11:18:33 ON 22 APR 2005
L28             0 S L24 NOT L26

     FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS' ENTERED AT 11:22:48 ON 22
     APR 2005
L29            59 S L24 NOT L26
L30            40 DUP REM L29 (19 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 11:23:32 ON 22 APR 2005

=> d cost
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CONNECT CHARGES                                         0.00     140.33
NETWORK CHARGES                                         3.00       7.02
DISPLAY CHARGES                                         0.00     124.15
                                                     -------    -------
FULL ESTIMATED COST                                     3.00     271.50

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                     0.00     -15.33

IN FILE 'STNGUIDE' AT 11:53:20 ON 22 APR 2005


=> file medline biosis hcaplus embase wpids
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     3.00     271.50

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                     0.00     -15.33

FILE 'MEDLINE' ENTERED AT 11:53:31 ON 22 APR 2005

FILE 'BIOSIS' ENTERED AT 11:53:31 ON 22 APR 2005
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FILE 'HCAPLUS' ENTERED AT 11:53:31 ON 22 APR 2005
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FILE 'WPIDS' ENTERED AT 11:53:31 ON 22 APR 2005
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=> d his

L1            2 S E4

     FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS' ENTERED AT 10:46:14 ON 22
     APR 2005
L2       624438 S (DIABETES(W)MELLITUS) OR "TYPE 1 DIABETES" OR "TYPE 2 DIABETE
L3       316701 S (MEDIUM(W)CHAIN(W)TRIGLYCERIDE?) OR TRIACYLGLYCEROL? OR TRIGL
L4       198146 S (OLEIC ACID?) OR "9-OCTADECENOIC ACID" OR OLEATE? OR (OLIVE O
L5       122681 S (LINOLEIC ACID?) OR LINOLEATE? OR "9,12-OCTADECADIENOIC ACID"
L6         2949 S (Ω)(W)"6"(W)FATTY(W)ACID? OR (DOUBLE(W)UNSATURATED(W)TR
L7        85651 S ((A)(W)LINOLENIC(W)ACID?) OR (LINOLENIC ACID?) OR LINOL
L8            1 S (TRIPLE(W)UNSATURATED(W)TRIGLYCERIDE?)
L9        52757 S EICOSAPENTAEN? OR EICOSAPENTAENOIC ACID? OR TIMNODONIC ACID?
L10       60428 S DOCOSAHEXAEN? OR DOCOSAHEXAENOIC ACID? OR "DHA" OR (FISH OIL?
L11        4405 S (SATURATED(W)LONG(W)CHAIN(W)(TRIGLYCERIDE? OR TRIACYLGLYCEROL
L12      152425 S EMULSIFIER OR EMULSIF?
L13       69467 S MONOGLYCERIDE? OR MONOACYLGLYCEROL? OR DIGLYCERIDE? OR DIACYL
L14      421736 S "VITAMIN A" OR RETINOL? OR RETINYL PALMITATE? OR "VITAMIN D"
L15       48454 S CAROTIN? OR (BETA(W)(CAROTIN? OR CAROTENE?)) OR ((B)(W)
L16       27388 S FLAVORING? OR (BUTTER(W)FLAVOR?) OR ROSEMARY? OR (ROSEMARY(W)
L17      130972 S RETINYL PALMITATE? OR "VITAMIN D3" OR CHOLECALCIFEROL? OR "VI
L18      197160 S "VITAMIN B6" OR PICOLINE? OR PYRIDOXINE? OR PYRIDOXAL? OR PYR
L19      404698 S "VITAMIN C" OR ASCORBIC ACID? OR ASCORBYL PALMITATE? OR "VITA
L20     4846639 S ZINC? OR CHROM? OR MANGANESE?
L21       32231 S L2 AND L3
L22         699 S L21 AND L4
L23         204 S L22 AND (L5 OR L6 OR L7 OR L8)
L24          73 S L23 AND (L9 OR L10)
L25           1 S L24 AND L11
L26          14 S L24 AND (L12 OR L13 OR L14 OR L15 OR L16)
L27          11 DUP REM L26 (3 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 11:18:33 ON 22 APR 2005
L28           0 S L24 NOT L26

     FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS' ENTERED AT 11:22:48 ON 22
     APR 2005
L29          59 S L24 NOT L26
L30          40 DUP REM L29 (19 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 11:23:32 ON 22 APR 2005

     FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS' ENTERED AT 11:53:31 ON 22
     APR 2005

=> s metabol? or (fat metabol?) or (lipid metabol?) or (fatty acid metabol?)
   2 FILES SEARCHED...
L31     7724971 METABOL? OR (FAT METABOL?) OR (LIPID METABOL?) OR (FATTY ACID
                METABOL?)

=> s l22 and l31
L32         453 L22 AND L31

=> s l23 and l31
L33         101 L23 AND L31

=> dup rem l33
PROCESSING COMPLETED FOR L33
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L34           80 DUP REM L33 (21 DUPLICATES REMOVED)
                 ANSWERS '1-26' FROM FILE MEDLINE
                 ANSWERS '27-36' FROM FILE BIOSIS

=> s l34 not l30
L35           62 L34 NOT L30

=> s l35 not l27
L36           56 L35 NOT L27

=> dup rem l36
PROCESSING COMPLETED FOR L36
L37           56 DUP REM L36 (0 DUPLICATES REMOVED)
                 ANSWERS '1-19' FROM FILE MEDLINE
                 ANSWERS '20-26' FROM FILE BIOSIS
                 ANSWERS '27-41' FROM FILE HCAPLUS
                 ANSWERS '42-54' FROM FILE EMBASE
                 ANSWERS '55-56' FROM FILE WPIDS

=> d l37 ibib ed abs

L37  ANSWER 1 OF 56     MEDLINE on STN
ACCESSION NUMBER:    2004396304     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 15298769
TITLE:               Protective role of Phaseolus vulgaris on changes in the
                     fatty acid composition in experimental diabetes.
AUTHOR:              Pari Leelavinothan; Venkateswaran Subramanian
CORPORATE SOURCE:    Department of Biochemistry, Faculty of Science, Annamalai
                     University, Annamalainagar-608 002, Tamil Nadu, India..
                     paribala@sancharnet.in
SOURCE:              Journal of medicinal food, (2004 Summer) 7 (2) 204-9.
                     Journal code: 9812512. ISSN: 1096-620X.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         200410
ENTRY DATE:          Entered STN: 20040810
                     Last Updated on STN: 20041008
                     Entered Medline: 20041007
ED   Entered STN: 20040810
     Last Updated on STN: 20041008
     Entered Medline: 20041007
AB   The present investigation was carried out to evaluate the effect of
     Phaseolus vulgaris, an indigenous plant used in Unani and Ayurvedic
     medicine in India, on blood glucose, plasma insulin, cholesterol,
     triglycerides, free fatty acids, phospholipids, and fatty acid
     composition of total lipids in liver, kidney, and brain of normal and
     streptozotocin (STZ) diabetic rats. The results show that there was a
     significant increase in tissue cholesterol, triglycerides, free
     fatty acids, and phospholipids in STZ diabetic rats. The analysis of
     fatty acids showed that there was a significant increase in the
     concentrations of palmitic acid (16:1), stearic acid (18:0), and
     oleic acid (18:1) in liver, kidney, and brain, whereas
     the concentrations of linolenic acid (18:3) and
     arachidonic acid (20:4) were significantly decreased. Oral administration
     of the aqueous extract of P. vulgaris pods (200 mg/kg of body weight) for
     45 days to diabetic rats decreased the concentrations of lipids and fatty
     acids, viz., palmitic, stearic, and oleic acids,
     whereas linolenic and arachidonic acids were elevated. Similarly, the
     administration of P. vulgaris pod extract (PPEt) to normal animals
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resulted in a significant hypolipidemic effect. These results suggest that PPEt exhibits hypoglycemic and hypolipidemic effects in STZ diabetic rats. It also prevents the fatty acid changes produced during [illegible]

```
=> d his

     (FILE 'HOME' ENTERED AT 10:45:15 ON 22 APR 2005)

     FILE 'HCAPLUS' ENTERED AT 10:45:27 ON 22 APR 2005
                E HEIRLER H/AU
L1              2 S E4

     FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS' ENTERED AT 10:46:14 ON 22
     APR 2005
L2         624438 S (DIABETES(W)MELLITUS) OR "TYPE 1 DIABETES" OR "TYPE 2 DIABETE
L3         316701 S (MEDIUM(W)CHAIN(W)TRIGLYCERIDE?) OR TRIACYLGLYCEROL? OR TRIGL
L4         198146 S (OLEIC ACID?) OR "9-OCTADECENOIC ACID" OR OLEATE? OR (OLIVE O
L5         122681 S (LINOLEIC ACID?) OR LINOLEATE? OR "9,12-OCTADECADIENOIC ACID"
L6           2949 S (Ω)(W)"6"(W)FATTY(W)ACID? OR (DOUBLE(W)UNSATURATED(W)TR
L7          85651 S ((A)(W)LINOLENIC(W)ACID?) OR (LINOLENIC ACID?) OR LINOL
L8              1 S (TRIPLE(W)UNSATURATED(W)TRIGLYCERIDE?)
L9          52757 S EICOSAPENTAEN? OR EICOSAPENTAENOIC ACID? OR TIMNODONIC ACID?
L10         60428 S DOCOSAHEXAEN? OR DOCOSAHEXAENOIC ACID? OR "DHA" OR (FISH OIL?
L11          4405 S (SATURATED(W)LONG(W)CHAIN(W)(TRIGLYCERIDE? OR TRIACYLGLYCEROL
L12        152425 S EMULSIFIER OR EMULSIF?
L13         69467 S MONOGLYCERIDE? OR MONOACYLGLYCEROL? OR DIGLYCERIDE? OR DIACYL
L14        421736 S "VITAMIN A" OR RETINOL? OR RETINYL PALMITATE? OR "VITAMIN D"
L15         48454 S CAROTIN? OR (BETA(W)(CAROTIN? OR CAROTENE?)) OR ((B)(W)
L16         27388 S FLAVORING? OR (BUTTER(W)FLAVOR?) OR ROSEMARY? OR (ROSEMARY(W)
L17        130972 S RETINYL PALMITATE? OR "VITAMIN D3" OR CHOLECALCIFEROL? OR "VI
L18        197160 S "VITAMIN B6" OR PICOLINE? OR PYRIDOXINE? OR PYRIDOXAL? OR PYR
L19        404698 S "VITAMIN C" OR ASCORBIC ACID? OR ASCORBYL PALMITATE? OR "VITA
L20       4846639 S ZINC? OR CHROM? OR MANGANESE?
L21         32231 S L2 AND L3
L22           699 S L21 AND L4
L23           204 S L22 AND (L5 OR L6 OR L7 OR L8)
L24            73 S L23 AND (L9 OR L10)
L25             1 S L24 AND L11
L26            14 S L24 AND (L12 OR L13 OR L14 OR L15 OR L16)
L27            11 DUP REM L26 (3 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 11:18:33 ON 22 APR 2005
L28             0 S L24 NOT L26

     FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS' ENTERED AT 11:22:48 ON 22
     APR 2005
L29            59 S L24 NOT L26
L30            40 DUP REM L29 (19 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 11:23:32 ON 22 APR 2005

     FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS' ENTERED AT 11:53:31 ON 22
     APR 2005
L31       7724971 S METABOL? OR (FAT METABOL?) OR (LIPID METABOL?) OR (FATTY ACID
L32           453 S L22 AND L31
L33           101 S L23 AND L31
L34            80 DUP REM L33 (21 DUPLICATES REMOVED)
L35            62 S L34 NOT L30
L36            56 S L35 NOT L27
L37            56 DUP REM L36 (0 DUPLICATES REMOVED)
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=> s l22 (L) l31
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L122 (L) L161'

PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L125 (L) L164'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L126 (L) L165'
L38        453 L22 (L) L31

=> d l37 1-56 ibib ed abs
THE ESTIMATED COST FOR THIS REQUEST IS 103.08 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:n
REQUEST CANCELED

=> d cost
COST IN U.S. DOLLARS                              SINCE FILE      TOTAL
                                                     ENTRY    SESSION
CONNECT CHARGES                                      13.75     154.08
NETWORK CHARGES                                       0.48       7.50
DISPLAY CHARGES                                       0.22     124.37
                                                   -------    -------
FULL ESTIMATED COST                                  14.45     285.95

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)        SINCE FILE      TOTAL
                                                     ENTRY    SESSION
CA SUBSCRIBER PRICE                                   0.00     -15.33

IN FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS'
AT 11:58:30 ON 22 APR 2005


=> d l37 1-56 ibib ed abs
THE ESTIMATED COST FOR THIS REQUEST IS 103.08 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:y

L37  ANSWER 1 OF 56     MEDLINE on STN
ACCESSION NUMBER:    2004396304     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 15298769
TITLE:               Protective role of Phaseolus vulgaris on changes in the
                     fatty acid composition in experimental diabetes.
AUTHOR:              Pari Leelavinothan; Venkateswaran Subramanian
CORPORATE SOURCE:    Department of Biochemistry, Faculty of Science, Annamalai
                     University, Annamalainagar-608 002, Tamil Nadu, India..
                     paribala@sancharnet.in
SOURCE:              Journal of medicinal food, (2004 Summer) 7 (2) 204-9.
                     Journal code: 9812512. ISSN: 1096-620X.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         200410
ENTRY DATE:          Entered STN: 20040810
                     Last Updated on STN: 20041008
                     Entered Medline: 20041007
ED   Entered STN: 20040810
     Last Updated on STN: 20041008
     Entered Medline: 20041007
AB   The present investigation was carried out to evaluate the effect of
     Phaseolus vulgaris, an indigenous plant used in Unani and Ayurvedic
     medicine in India, on blood glucose, plasma insulin, cholesterol,
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triglycerides, free fatty acids, phospholipids, and fatty acid composition of total lipids in liver, kidney, and brain of normal and streptozotocin (STZ) diabetic rats. The results show that there ...

oleic acid (18:1) in liver, kidney, and brain, whereas the concentrations of **linolenic acid** (18:3) and arachidonic acid (20:4) were significantly decreased. Oral administration of the aqueous extract of P. vulgaris pods (200 mg/kg of body weight) for 45 days to diabetic rats decreased the concentrations of lipids and fatty acids, viz., palmitic, stearic, and **oleic acids**, whereas linolenic and arachidonic acids were elevated. Similarly, the administration of P. vulgaris pod extract (PPEt) to normal animals resulted in a significant hypolipidemic effect. These results suggest that PPEt exhibits hypoglycemic and hypolipidemic effects in STZ diabetic rats. It also prevents the fatty acid changes produced during diabetes. The effect of PPEt at 200 mg/kg of body weight was better than that of glibenclamide.

L37 ANSWER 2 OF 56 MEDLINE on STN
ACCESSION NUMBER: 2002496260 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12138107
TITLE: **Oleate** and **linoleate** enhance the growth-promoting effects of insulin-like growth factor-I through a phospholipase D-dependent pathway in arterial smooth muscle cells.
AUTHOR: Askari Bardia; Carroll Mairead A; Capparelli Maria; Kramer Farah; Gerrity Ross G; Bornfeldt Karin E
CORPORATE SOURCE: Department of Pathology, University of Washington School of Medicine, Seattle, Washington 98195, USA.
CONTRACT NUMBER: HL07312 (NHLBI)
HL25394 (NHLBI)
HL34300 (NHLBI)
HL55789 (NHLBI)
HL62887 (NHLBI)
SOURCE: Journal of biological chemistry, (2002 Sep 27) 277 (39) 36338-44. Electronic Publication: 2002-07-22.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200211
ENTRY DATE: Entered STN: 20021003
Last Updated on STN: 20030105
Entered Medline: 20021113
ED Entered STN: 20021003
Last Updated on STN: 20030105
Entered Medline: 20021113
AB Diabetes causes accelerated atherosclerosis and subsequent cardiovascular disease through mechanisms that are poorly understood. We have previously shown, using a porcine model of diabetes-accelerated atherosclerosis, that diabetes leads to an increased accumulation and proliferation of arterial smooth muscle cells in atherosclerotic lesions and that this is associated with elevated levels of plasma **triglycerides**. We therefore used the same model to investigate the mechanism whereby diabetes may stimulate smooth muscle cell proliferation. We show that lesions from diabetic pigs fed a cholesterol-rich diet contain abundant insulin-like growth factor-I (IGF-I), in contrast to lesions from non-diabetic pigs. Furthermore, two fatty acids common in **triglycerides**, **oleate** and **linoleate**, enhance the growth-promoting effects of IGF-I in smooth muscle cells isolated from these animals. These fatty acids accumulate

predominantly in the membrane phospholipid pool; **oleate** accumulates preferentially in phosphatidylcholine and phosphatidylethanolamine, whereas linoleate is found mainly in

linoleate in lesions may contribute to accumulation and proliferation of smooth muscle cells and lesion progression in diabetes-accelerated atherosclerosis.

L37 ANSWER 3 OF 56 MEDLINE on STN
ACCESSION NUMBER: 2002020949 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11453041
TITLE: Postprandial **triglycerides** and endothelial function.
AUTHOR: Jagla A; Schrezenmeir J
CORPORATE SOURCE: Institute of Physiology and Biochemistry of Nutrition, Federal Research Centre Kiel, Germany.
SOURCE: Experimental and clinical endocrinology & diabetes : official journal, German Society of Endocrinology [and] German Diabetes Association, (2001) 109 (4) S533-47. Ref: 149
Journal code: 9505926. ISSN: 0947-7349.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20020121
Last Updated on STN: 20020121
Entered Medline: 20011205

ED Entered STN: 20020121
Last Updated on STN: 20020121
Entered Medline: 20011205

AB Several studies support the association between postprandially elevated **triglyceride** levels and atherosclerosis. Histological and cell culture investigations revealed, that **triglyceride** rich postprandial lipoproteins are taken up by macrophages and smooth muscle cells and are detectable as part of foam cells in vascular lesions. Remnant particles, generated by lipolysis of postprandial lipoproteins in vitro and fatty acids increase the permeability of the endothelium and are cytotoxic for endothelial cells. Besides these morphological changes of cells, lipoproteins have been shown to exert effects on cellular functions like the expression of membrane proteins and the production or release of several bioactive substances regulating communication with blood cells and other cell systems of the vascular wall, blood pressure and hemostasis. This review concentrates on the influence of postprandial lipoproteins on factors involved in the interaction of endothelial cells with blood leukocytes and factors mediating blood pressure regulation. Increased expression of adhesion molecules has been detected immunehistochemically in atherosclerotic plaques in animals and humans. It was demonstrated that patients with elevated **triglyceride** levels have increased levels of soluble adhesion molecules. Furthermore, postprandial lipoproteins were shown to induce membrane expression of adhesion molecules. This effect seems to be at least in part mediated by the oxidative modification of the particles. Accordingly chylomicrons separated after ingestion of safflower oil, rich in polyunsaturated **linoleic acid**, induced higher adhesion molecule expression at higher oxidant concentration compared with chylomicrons separated after ingestion of **olive oil**, rich in monounsaturated **oleic acid**. Several authors described

effects of fatty acids on the expression of adhesion molecules. On the one hand, they may exert stimulatory effects as such, on the other hand cytokine-induced adhesion molecule expression may be inhibited

increasingly attention in the last two decades and is regarded as protective against hypertension and atherosclerosis. It was demonstrated that chylomicrons and their remnants inhibited endothelium-dependent relaxations in isolated aortas. Vasodilatatory responses and nitric oxide **metabolism** were shown to be affected by the amount and composition of dietary fat. Cell culture experiments revealed modulation of NO release by certain fatty acids. Plasma levels of endothelin-1, a strong vasoconstrictor, have been shown to be increased in patients with **type 2 diabetes** and **metabolic** syndrome, respectively. Postprandially elevated **triglycerides** increased endothelin-levels in addition to insulin in patients with **metabolic** syndrome. In summary, there is evidence that the association between postprandial **triglycerides** and the **metabolic** syndrome is driven by direct influences on endothelial functions because plasma **triglyceride** levels are associated with levels of humoral risk markers of endothelial origin, and postprandial lipoproteins stimulate the release and/or expression of endothelial mediators in vitro, which induce atherogenesis and hypertension.

L37 ANSWER 4 OF 56 MEDLINE on STN
ACCESSION NUMBER: 2003129429 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12643112
TITLE: Dietary fats and **diabetes mellitus**: is there a good fat?.
AUTHOR: Segal-Isaacson C J; Carello E; Wylie-Rosett J
CORPORATE SOURCE: Albert Einstein College of Medicine of Yeshiva University, Department of Epidemiology and Social Medicine, Belfer 1308D, 1300 Morris Park Avenue, Bronx, NY 10461, USA.. isaacson@aecom.yu.edu
SOURCE: Current diabetes reports, (2001 Oct) 1 (2) 161-9. Ref: 48
Journal code: 101093791. ISSN: 1534-4827.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200304
ENTRY DATE: Entered STN: 20030320
Last Updated on STN: 20030406
Entered Medline: 20030404
ED Entered STN: 20030320
Last Updated on STN: 20030406
Entered Medline: 20030404
AB As knowledge of the fatty acid functions has increased, so has the complexity of making dietary fat recommendations to people with **type 2 diabetes**. **Oleic acid** seems to offer a slight advantage over **linoleic acid** in reducing plasma glucose, insulin levels, total cholesterol, low-density lipoproteins (LDLs), and **triglycerides**, but may also have atherogenic properties through another mechanism. A diet containing a higher proportion of polyunsaturated fatty acids (PUFAs) may require a concomitant increase in antioxidant intake because PUFAs oxidize easily and are then converted to oxidized LDL, which is more atherogenic. In addition to raising total and LDL cholesterol, long chain saturated free fatty acids may interact with plasma glucose to increase insulin secretion. **Omega-3 fatty acids** decrease **triglycerides** and reduce the risk of fatal cardiac

arrhythmias. Glycemic control does not appear to be adversely affected by **omega-3 fatty acids** at amounts of up to 3 g/d

TITLE: Dietary unsaturated fatty acids in **type 2 diabetes**: higher levels of postprandial lipoprotein on a **linoleic acid**-rich **sunflower oil** diet compared with an **oleic acid**-rich **olive oil** diet.
AUTHOR: Madigan C; Ryan M; Owens D; Collins P; Tomkin G H
CORPORATE SOURCE: Department of Clinical Medicine, Trinity College.
SOURCE: Diabetes care, (2000 Oct) 23 (10) 1472-7.
Journal code: 7805975. ISSN: 0149-5992.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010202
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010202
AB OBJECTIVE: The present study was undertaken to examine the effect of a polyunsaturated fat diet compared with an isocaloric Mediterranean-style monounsaturated fat diet. RESEARCH DESIGN AND METHODS: This was a randomized 2-week crossover study on either a high-polyunsaturated or a high-monounsaturated fat diet in 11 well-controlled diabetic men. Blood was taken fasting and for up to 8 h after a high fat meal. Lipoproteins were isolated by sequential ultracentrifugation. Apolipoprotein (apo) B48 and apo B100 were separated by PAGE. Fatty acids were analyzed by gas-liquid chromatography RESULTS: Fasting blood glucose and insulin levels were significantly higher on the **linoleic acid** diet compared with the **oleic acid** diet ($P < 0.01$ and $P < 0.002$, respectively). Plasma cholesterol and LDL cholesterol levels were also significantly higher on the **linoleic acid** diet ($P < 0.001$). Likewise, fasting chylomicron apo B48 and apo B100 ($P < 0.05$) and postprandial chylomicron and VLDL apo B48 and B100 ($P < 0.05$) were also higher on the **linoleic acid** diet. CONCLUSIONS: This study suggests that, in **type 2 diabetes**, an **oleic acid**-rich Mediterranean-type diet versus a **linoleic acid**-enriched diet may reduce the risk of atherosclerosis by decreasing the number of chylomicron remnant particles.

L37 ANSWER 6 OF 56 MEDLINE on STN
ACCESSION NUMBER: 2001034185 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11063437
TITLE: Diet-induced change in fatty acid composition of plasma **triacylglycerols** is not associated with change in glucagon-like peptide 1 or insulin sensitivity in people with **type 2 diabetes**.
AUTHOR: Brynes A E; Edwards C M; Jadhav A; Ghatei M A; Bloom S R; Frost G S
CORPORATE SOURCE: Nutrition and Dietetic Research Group, the Endocrine Unit, and the Lipoprotein Team, Imperial College School of Medicine, Hammersmith Hospital, London.

SOURCE: American journal of clinical nutrition, (2000 Nov) 72 (5) 1111-8.
Journal code: 0376027. ISSN: 0002-9165

LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001130

ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001130

AB BACKGROUND: Polyunsaturated fatty acids (PUFAs) and **monounsaturated fatty acids** (**MUFAs**) have been shown to positively affect blood lipids; however, their comparative effects on insulin sensitivity are unclear. OBJECTIVE: Our objective was to investigate whether chronic intake of **MUFAs** or PUFAs improves insulin sensitivity in people with **type 2 diabetes** via stimulation of the endogenous gut hormone glucagon-like peptide 1 [7-36] amide (GLP-1). DESIGN: Nine overweight people with **type 2 diabetes** received isoenergetic high-**MUFA** (20.3 +/- 3.5% of total energy) or high-PUFA (13.4 +/- 1. 3%) diets for 24 d in a randomized, double-blind crossover design. RESULTS: Weight and glycemic control remained stable throughout the study. Despite a significant change in the plasma **triacylglycerol** linoleic-**oleic acid** ratio (L:O) with both diets (**MUFA**: from 0.46 +/- 0.03 to 0.29 +/- 0.02, P: < 0.005; PUFA: from 0.36 +/- 0.04 to 0.56 +/- 0.05, P: < 0.05) and the phospholipid L:O (1.7 +/- 0.1 to 2.0 +/- 0.3; P: = 0.04) during consumption of the PUFA diet, this change was not associated with a change in insulin sensitivity, measured by the short-insulin-tolerance test. There was a significant reduction in the ratio of total to HDL cholesterol during consumption of the PUFA diet (5.2 +/- 0.4 compared with 4.7 +/- 0.3; P: = 0.005) but no change with the **MUFA** diet. There was no change in the fasting or postprandial incremental area under the curve in response to an identical standard test meal for glucose, insulin, **triacylglycerol**, nonesterified fatty acids, or GLP-1. CONCLUSIONS: Over the 3-wk intervention period, diet-induced change in the **triacylglycerol** or phospholipid L:O was not associated with either increased stimulation of GLP-1 or a change in insulin sensitivity in people with **type 2 diabetes**.

L37 ANSWER 7 OF 56 MEDLINE on STN
ACCESSION NUMBER: 96375451 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8781762
TITLE: Lipoprotein composition in NIDDM: effects of dietary **oleic acid** on the composition, oxidisability and function of low and high density lipoproteins.
AUTHOR: Dimitriadis E; Griffin M; Collins P; Johnson A; Owens D; Tomkin G H
CORPORATE SOURCE: Department of Clinical Medicine, Trinity College Dublin, Ireland.
SOURCE: Diabetologia, (1996 Jun) 39 (6) 667-76.
Journal code: 0006777. ISSN: 0012-186X.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199612

ENTRY DATE: Entered STN: 19970128
Last Updated on STN: 19970128
Entered Medline: 19961205

pathogenesis of atherosclerosis and is related to the fatty acid composition which is altered in **diabetes mellitus**. This study examines the relationship between the fatty acid composition of LDL and high density lipoprotein (HDL) and lipoprotein oxidation. A group of nine non-insulin-dependent diabetic (NIDDM) patients were compared to seven healthy control subjects before and after a high monounsaturated diet. Lipoproteins were isolated and oxidisability was measured by conjugated diene formation and lipid peroxide analysis. Serum HDL cholesterol was significantly lower in the diabetic patients. LDL cholesteryl ester **linoleic acid** in the diabetic patients was significantly higher at baseline and decreased after diet ($p < 0.05$) while **oleic acid** increased in both diabetic and non-diabetic subjects ($p < 0.05$). HDL cholesteryl ester **oleic acid** was lower in the diabetic patients compared with control subjects ($p < 0.05$) before diet and it increased significantly after diet ($p < 0.05$). LDL lipid peroxides and conjugated diene formation were related to LDL glycation ($r = 0.46$, $p < 0.05$ and $r = 0.49$, $p < 0.05$, respectively). Both decreased following diet (lipid peroxides for diabetic patients from 476 +/- 30 to 390 +/- 20 nmol/mg protein $p < 0.05$ and for control subjects from 350 +/- 36 to 198 +/- 30 nmol/mg protein $p < 0.05$). HDL conjugated diene formation decreased in both groups after diet but only significantly in the control group (55.4 +/- 7.5 to 53.2 +/- 6.7 nmol/mg protein for diabetic patients and 45.8 +/- 6.4 to 31.6 +/- 4.8 nmol/mg protein $p < 0.05$ for control subjects). There was a positive correlation between LDL lipid peroxide formation and percentage of cholesteryl ester **linoleic acid** in LDL from diabetic patients ($r = 0.61$, $p < 0.05$) and control subjects ($r = 0.91$, $p < 0.01$). Fatty acid composition of LDL was reflected in the composition of HDL. In the presence of HDL lipoprotein peroxidation decreased. This decrease in lipoprotein peroxidation was positively related to the percentage of **linoleic acid** in LDL ($r = 0.71$, $p < 0.05$). This study confirms the close relationship between the fatty acid composition of LDL and HDL and demonstrates the importance of the fatty acid composition of the cholesteryl ester fraction in relation to LDL oxidation in diabetes. **Linoleic acid** in HDL appears to be a protecting factor against oxidation.

L37 ANSWER 8 OF 56 MEDLINE on STN
ACCESSION NUMBER: 96037100 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7587908
TITLE: Effects of insulin therapy upon plasma lipid fatty acids and platelet aggregation in NIDDM with secondary failure to oral antidiabetic agents.
AUTHOR: Rodier M; Colette C; Gouzes C; Michel F; Crastes de Paulet A; Monnier L
CORPORATE SOURCE: Department of Internal Medicine T, Hospital Caremeau, Nimes, France.
SOURCE: Diabetes research and clinical practice, (1995 Apr) 28 (1) 19-28.
Journal code: 8508335. ISSN: 0168-8227.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199512

ENTRY DATE: Entered STN: 19960124
Last Updated on STN: 19980206
Entered Medline: 19951207

fatty acid metabolism and platelet function were studied in 18 non-obese non-insulin-dependent diabetics (NIDDs) with secondary failure to oral antidiabetic drugs (OAD). Patients were randomly allocated either to continue maximal OAD (Group I, n = 9) or to receive a multiple injection regimen of insulin therapy (Group II, n = 9) for a 6-month period. At baseline both groups were identical for clinical and biological parameters. At study day 180, fasting blood glucose ($P < 0.01$) and mean capillary blood glucose ($P < 0.05$) were reduced in group II but the difference between HbA1 percentages remained non-significant. At study day 60, in total plasma lipids, **oleic acid** was lower ($P < 0.05$), **linoleic acid** ($P < 0.05$) and the sum of polunsaturated fatty acids (PUFA) ($P < 0.05$) were higher in group II than I. In **triglycerides**, palmitic acid was lower in group II at study days 60 ($P < 0.01$) and 180 ($P < 0.05$), whereas gamma-**linolenic acid** was decreased ($P < 0.05$) at study day 180 only. A similar change was noted in cholesterol esters for gamma-**linolenic acid** at study day 60 ($P < 0.05$). No difference was noted between both groups for platelet agregation, insulin sensitivity and clinical parameters despite a significant increase in body weight in group II at study day 180. Positive correlations were obtained between the content of different lipid fractions in some PUFA and the glucose clearance. We conclude that optimized insulin therapy in NIDDs with secondary failure to OAD leads to a transient improvement in glucidic and lipidic **metabolism** but has no significant effect upon platelet aggregation and insulin sensitivity.

L37 ANSWER 9 OF 56 MEDLINE on STN
ACCESSION NUMBER: 94276678 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7911870
TITLE: Fatty acid composition of erythrocytes and plasma **triglyceride** and cardiovascular risk in Asian diabetic patients.
COMMENT: Comment in: Lancet. 1994 Jun 18;343(8912):1518. PubMed ID: 7911867
Comment in: Lancet. 1994 Oct 8;344(8928):1030. PubMed ID: 7999151
Comment in: Lancet. 1994 Oct 8;344(8928):1030-1. PubMed ID: 7934417
AUTHOR: Peterson D B; Fisher K; Carter R D; Mann J
CORPORATE SOURCE: Diabetes Research Laboratories, Radcliffe Infirmary, Oxford, UK.
SOURCE: Lancet, (1994 Jun 18) 343 (8912) 1528-30.
Journal code: 2985213R. ISSN: 0140-6736.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199407
ENTRY DATE: Entered STN: 19940729
Last Updated on STN: 19960129
Entered Medline: 19940715
ED Entered STN: 19940729
Last Updated on STN: 19960129
Entered Medline: 19940715
AB The excess of coronary heart disease in Indian Asians compared with Europeans is unexplained by conventional risk factors, although the high prevalence of diabetes may play a part. To explore the contribution of

diet we compared the fatty acid composition of erythrocyte membrane phospholipid and plasma **triglyceride** in 36 Gujarati Asians and 24 Europeans with non-insulin-dependent diabetes. Erythrocytes from Asian

oleic acid (18:1n-9) in erythrocytes was 16.7 (0.2) in Asians and 20.5 (0.6) in Europeans ($p = 0.0001$), and total n-6:n-3 ratio was, respectively, 12.8 (0.7) and 6.7 (0.7) ($p = 0.0001$). A high dietary intake of **linoleic acid** may not be cardioprotective unless balanced by significant intakes of oleic and n-3 series fatty acids, at least in diabetic Indian Asians. By itself, the conventional recommendation to substitute polyunsaturated for saturated fat in the diet may be inadequate to reduce thrombogenesis, and the overall balance of fatty acids, including monounsaturates, should be considered.

L37 ANSWER 10 OF 56 MEDLINE on STN
ACCESSION NUMBER: 95080490 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7988779
TITLE: Serum cholesterol and cholesterol and lipoprotein **metabolism** in hypercholesterolaemic NIDDM patients before and during sitostanol ester-margarine treatment.
AUTHOR: Gylling H; Miettinen T A
CORPORATE SOURCE: Second Department of Medicine, University of Helsinki, Finland.
SOURCE: Diabetologia, (1994 Aug) 37 (8) 773-80.
Journal code: 0006777. ISSN: 0012-186X.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: (CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199501
ENTRY DATE: Entered STN: 19950124
Last Updated on STN: 19950124
Entered Medline: 19950110
ED Entered STN: 19950124
Last Updated on STN: 19950124
Entered Medline: 19950110
AB Cholesterol absorption and **metabolism** and LDL and HDL kinetics were investigated in 11 hypercholesterolaemic non-insulin-dependent diabetic men off and on a hypolipidaemic treatment with sitostanol ester, (3 g sitostanol daily) dissolved in **rapeseed oil** margarine, by a double-blind crossover study design. Serum total, VLDL and LDL cholesterol and apoprotein B fell significantly by 6 +/- 2, 12 +/- 6, 9 +/- 3 and 6 +/- 2%, mean +/- SEM, and HDL cholesterol was increased by 11 +/- 4% ($p < 0.05$) by sitostanol ester. LDL cholesterol and apoprotein B were significantly decreased in the dense (1.037-1.055 g/ml), but not light, LDL subfraction due to a significantly diminished transport rate for LDL apoprotein B, while the fractional catabolic rate was unchanged. HDL kinetics, measured with autologous apoprotein A I, was unaffected by sitostanol ester. Cholesterol absorption efficiency was markedly reduced from 25 +/- 2 to 9 +/- 2% ($p < 0.001$) during sitostanol ester followed by proportionately decreased serum plant sterol proportions. Cholesterol precursor sterol proportions in serum, fecal neutral sterol excretion, and cholesterol synthesis, cholesterol transport, and biliary secretion were all significantly increased by sitostanol ester. We conclude that the sitostanol ester-induced decrease in cholesterol absorption compensatorily stimulated cholesterol synthesis, had no effect on fractional catabolic rate, but decreased transport rate for LDL apoprotein B so that serum total, VLDL and LDL cholesterol levels were decreased.(ABSTRACT TRUNCATED AT 250 WORDS)

L37 ANSWER 11 OF 56 MEDLINE on STN
ACCESSION NUMBER: 95009391 MEDLINE

AUTHOR: Seigneur M; Freyburger G; Gin H; Claverie M; Lardeau D; Lacape G; Le Moigne F; Crockett R; Boisseau M R
CORPORATE SOURCE: Laboratoire d'Hemobiologie, Hopital Cardiologique, Pessac-Bordeaux, France.
SOURCE: Diabetes research and clinical practice, (1994 Apr) 23 (3) 169-77.
Journal code: 8508335. ISSN: 0168-8227.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 19941222
Last Updated on STN: 19941222
Entered Medline: 19941027
ED Entered STN: 19941222
Last Updated on STN: 19941222
Entered Medline: 19941027
AB Fatty acid profiles of various lipid fractions were determined in carefully selected insulin-dependent and non-insulin-dependent diabetics to assess relationships between serum fatty acid composition and type of diabetes. Clear-cut hypertriglyceridemia with slight hypercholesterolemia was found in both diabetic types. The decrease of lignoceric acid in sphingomyelin is the only alteration found in both types of diabetes. In the insulin-dependent diabetics, there were increases in levels of **oleic acid** and of **alpha-linolenic acid** in esterified cholesterol, and in levels of **alpha-linolenic acid** in the **triglyceride** fraction. In the non-insulin-dependent diabetics, there were increases in levels of **oleic acid** and total **monounsaturated fatty acids** in the **triglyceride** fraction and there was an increase in levels of saturated fatty acids and a decrease in levels of polyunsaturated acids in phosphatidylcholine; in sphingomyelin, dihomogamma-**linoleic acid** levels were enhanced. Arachidonic acid levels were normal in our patient population.

L37 ANSWER 12 OF 56 MEDLINE on STN
ACCESSION NUMBER: 88035456 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3117998
TITLE: Soybean oil emulsion administration during parenteral nutrition in the preterm infant: effect on essential fatty acid, lipid, and glucose **metabolism**.
AUTHOR: Cooke R J; Yeh Y Y; Gibson D; Debo D; Bell G L
CORPORATE SOURCE: Department of Pediatrics, University of Tennessee, Memphis.
SOURCE: Journal of pediatrics, (1987 Nov) 111 (5) 767-73.
Journal code: 0375410. ISSN: 0022-3476.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198712
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 19980206
Entered Medline: 19871214
ED Entered STN: 19900305

Last Updated on STN: 19980206
Entered Medline: 19871214
AB To examine the effect of a soybean oil emulsion on essential fatty acid

phospholipid **linoleate** (percent and micrograms per milliliter) increased, the increase being greater in group 2. In both groups, percent content of arachidonate and 5,8,11-eicosatrienoate decreased, and that of **oleate** remained unchanged. In contrast, absolute content of arachidonate and **oleate** tended to increase, and that of 5,8,11-eicosatrienoate remained unchanged. At a lipid intake of 0.5 g/kg/d, no infants had hyperlipemia. When lipid intake exceeded 1.0 g/kg/d, the frequency of hypertriglyceridemia (**triglycerides** greater than 200 mg/dL) and free fatty acidemia, with the free fatty acid/molar albumin ratio exceeding 6:1, increased. Plasma glycerol increased slightly, but was substantially less than the rise in enzymatically determined **triglycerides**. **Hyperglycemia** was self-limiting and did not require alteration in dextrose intake. Thus, (1) infusion of a soybean oil emulsion at 0.5 to 2.0 g/kg/d maintains essential fatty acid status and phospholipid arachidonate concentrations; (2) significant hyperlipemia occurs when lipid intake exceeds 1.0 g/kg/d; (3) **hyperglycemia** associated with lipid infusion tends to be self-limiting and may not require alteration in lipid or dextrose intake; and (4) enzymatically determined **triglycerides** may be used to monitor lipid tolerance, provided that allowance is made for a small but systematic overestimation resulting from the rise in plasma glycerol.

L37 ANSWER 13 OF 56 MEDLINE on STN
ACCESSION NUMBER: 86242813 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3718739
TITLE: [Correlations between unsaturated fatty acids and medium C-chain **triglycerides**].
Correlazioni tra acidi grassi insaturi e trigliceridi a media C-catena.
AUTHOR: Caponnetto A; Pagano M A; Rondinone R; Zunin P
SOURCE: Bollettino della Societa italiana di biologia sperimentale, (1986 Feb 28) 62 (2) 249-56.
Journal code: 7506962. ISSN: 0037-8771.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Italian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198607
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19980206
Entered Medline: 19860725
ED Entered STN: 19900321
Last Updated on STN: 19980206
Entered Medline: 19860725

L37 ANSWER 14 OF 56 MEDLINE on STN
ACCESSION NUMBER: 84183360 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6425101
TITLE: Increased arachidonic acid incorporation into platelet phospholipids in type 2 (non-insulin-dependent) diabetes.
AUTHOR: Takahashi R; Morita I; Saito Y; Ito H; Murota S
SOURCE: Diabetologia, (1984 Feb) 26 (2) 134-7.
Journal code: 0006777. ISSN: 0012-186X.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 198406

Entered Medline: 19840618

AB Arachidonic acid uptake activity was measured in platelets obtained from 27 Type 2 diabetic patients and 18 age-matched control subjects. In both groups after 1 h incubation almost all the incorporated 14C-arachidonic acid was located in the phospholipids of the platelets. Arachidonic acid was predominantly incorporated into phosphatidylcholine. The radioactivity incorporated into platelets increased linearly with incubation time, up to 90 min. The linear increase was observed at arachidonic acid concentrations of 0.1-1.0 micrograms/ml in both groups. The rate of incorporation of radioactivity in diabetic platelets was about 1.4 times higher than that in control platelets at all arachidonic acid concentrations studied. The arachidonic acid uptake activity of diabetic platelets (577 +/- 26 ng/60 min per 10(9) platelets) was significantly higher than that in control platelets (410 +/- 26 ng/60 min per 10(9) platelets). No significant correlations were found between the arachidonic acid uptake activity and fasting plasma glucose, total cholesterol or **triglyceride** levels. The arachidonic acid uptake activity of platelets was significantly higher in diabetic patients with proliferative retinopathy than in those with little or no background retinopathy. In addition, there were no significant differences between control and diabetic subjects in the uptake activity of platelets for **linoleic acid** and **oleic acid**. These data may explain the elevated arachidonic acid content in diabetic platelet phospholipids and enhancement of thromboxane synthesis in diabetes.

L37 ANSWER 15 OF 56 MEDLINE on STN
ACCESSION NUMBER: 84132871 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6230269
TITLE: Plasma lipid fatty acids and platelet function in insulin-dependent diabetic patients.
AUTHOR: Monnier L H; Chaintreuil J S; Colette C; Blotman M J; Crastes de Paulet P; Orsetti A; Crastes de Paulet A
SOURCE: Diabete & metabolisme, (1983 Dec) 9 (4) 283-7. Journal code: 7604157. ISSN: 0338-1684.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198404
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19980206
Entered Medline: 19840424

ED Entered STN: 19900319
Last Updated on STN: 19980206
Entered Medline: 19840424

AB Platelet function, estimated from plasma beta-thromboglobulin (beta-TG, ng/ml), is frequently altered in insulin-dependent diabetics (IDDs). As several factors may affect beta-TG, we studied respectively in 15 IDDs, the roles played by: (i) diabetic control evaluated from glycosylated haemoglobin (HbA1); (ii) plasma C-peptide and pancreatic glucagon; (iii) plasma lipids and the relative percentages of fatty acids in total plasma lipids. Plasma beta-TG did not correlate significantly with the first 3 parameters. However, beta-TG was correlated: (i) positively with plasma **triglycerides** (P less than 0.01), cholesterol (P less than 0.02), phospholipids (P less than 0.05) and total plasma lipids (P less than 0.01) and the percentage of **oleic acid** (C18 : 1 omega

9) in plasma lipids (P less than 0.01); (ii) negatively with the percentage of **linoleic acid** (C18 : 2 omega 6) in plasma lipids (P less than 0.02). No correlation was found between...

therapeutic measures which lower plasma lipids and increase the percentage of the **linoleic acid** in the body.

L37 ANSWER 16 OF 56 MEDLINE on STN
ACCESSION NUMBER: 75113297 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4375642
TITLE: The fatty acid pattern of adipose tissue and liver **triglycerides** according to fat droplet size in liver parenchymal cells of diabetic subjects.
AUTHOR: Singer P; Gnauck G; Honigmann G; Stolz P; Schliack V; Kettler L H; Buntrock P; Thoelke H
SOURCE: Diabetologia, (1974 Oct) 10 (5) 455-8.
Journal code: 0006777. ISSN: 0012-186X.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197505
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19750529
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19750529

L37 ANSWER 17 OF 56 MEDLINE on STN
ACCESSION NUMBER: 75074997 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4442359
TITLE: [Behavior of the fatty acid pattern of **triglycerides** in liver parenchyma and depot fats in diabetic patients in relation to fatty liver]. Zum Verhalten des Fettsauremusters der Triglyzeride im Leberparenchym und Depotfett bei Diabetikern in Abhangigkeit von der Leberverfettung.
AUTHOR: Singer P; Gnauck G; Thoelke H; Honigmann G; Buntrock P; Kettler L H
SOURCE: Deutsche Zeitschrift fur Verdauungs- und Stoffwechselkrankheiten, (1974) 34 (1) 27-35.
Journal code: 0372760. ISSN: 0012-1053.
PUB. COUNTRY: GERMANY, EAST: German Democratic Republic
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197504
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19750414
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19750414

L37 ANSWER 18 OF 56 MEDLINE on STN
ACCESSION NUMBER: 73203775 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4268280
TITLE: Effect of dietary glycerol on the (I- 14 C)stearic acid and (I- 14 (I- 14 C)**linoleic acid** desaturation of normal and diabetic rats.

AUTHOR: de Tomas M E; Peluffo R O; Mercuri O
SOURCE: Biochimica et biophysica acta, (1973 May 24) 306 (2) 149-55

FILE SEGMENT: Priority Journals
ENTRY MONTH: 197308
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19730823
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19730823

L37 ANSWER 19 OF 56 MEDLINE on STN
ACCESSION NUMBER: 74010818 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4745271
TITLE: [Hyperglyceridemia in juvenile and adult diabetes. Contribution to the study of **triglycerides** derived from **oleic acid** and **linoleic acid**].
L'hyperglyceridemie du diabete juvenile et de l'age mur. Contribution a l'etude des **triglycerides** derives de l'acide oleique et linoleique.
AUTHOR: Yanez-Polo M A
SOURCE: Annales d'endocrinologie, (1973 Mar-Apr) 34 (2) 107-13.
Journal code: 0116744. ISSN: 0003-4266.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197312
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19731216
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19731216

L37 ANSWER 20 OF 56 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:404501 BIOSIS
DOCUMENT NUMBER: PREV200300404501
TITLE: Comparisons of glucose and **lipid metabolism** in rats fed diacylglycerol and **triacylglycerol** oils.
AUTHOR(S): Sugimoto, Tomomi; Kimura, Tomoe; Fukuda, Hitomi; Iritani, Nobuko [Reprint Author]
CORPORATE SOURCE: Faculty of Human and Culture Studies, Tezukayama Gakuin University, 4-2-2 Harumidai, Sakai, Osaka, 590-0113, Japan iritani@hcs.tezuka-gu.ac.jp
SOURCE: Journal of Nutritional Science and Vitaminology, (February 2003) Vol. 49, No. 1, pp. 47-55. print.
ISSN: 0301-4800 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 3 Sep 2003
Last Updated on STN: 3 Sep 2003
ED Entered STN: 3 Sep 2003
Last Updated on STN: 3 Sep 2003
AB The effects of dietary 1,3-diacylglycerol-rich oil (DG oil) on biochemical findings related to glucose and **lipid metabolisms** were

investigated in comparison with **triacylglycerol** oil (TG oil) in normal rats. Young (7 wk-old) and old (8 mo-old) rats were fed a synthetic diet containing 10% (by weight) DG or TG oil for 1, 4, 8, or 12

groups, except that the plasma **triacylglycerol** concentrations were rather lower only in the portal vein of rats fed DG oil. The plasma glucose and free fatty acid concentrations were significantly higher in rats fed DG oil as compared to TG oil. In the old rats fed DG oil for 8 wk, the fasted plasma glucose and insulin concentrations were elevated and **glucose intolerance** was observed. The insulin receptor expression was not different due to dietary oil, but was markedly reduced with aging. Thus, the anti-obesity and lipid-lowering effects of dietary DG oil were not found. Moreover, it appeared that the **glucose intolerance** might be induced by dietary DG oil, particularly in the old rats.

L37 ANSWER 21 OF 56 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:470816 BIOSIS
DOCUMENT NUMBER: PREV200200470816
TITLE: Effects of alpha lipoic acid, ascorbic acid-6-palmitate, and fish oil on the glutathione, malonaldehyde, and fatty acids levels in erythrocytes of streptozotocin induced diabetic male rats.
AUTHOR(S): Yilmaz, Okkes [Reprint author]; Ozkan, Yusuf; Yildirim, Mehmet; Ozturk, A. Ihsan; Ersan, Yasemin
CORPORATE SOURCE: Department of Biology, Faculty of Science, Firat University, 23169, Elazig, Turkey
oyilmaz@firat.edu.tr
SOURCE: Journal of Cellular Biochemistry, (2002) Vol. 86, No. 3, pp. 530-539. print.
CODEN: JCEBD5. ISSN: 0730-2312.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Sep 2002
Last Updated on STN: 4 Sep 2002

ED Entered STN: 4 Sep 2002
Last Updated on STN: 4 Sep 2002

AB In this research, it has been aimed to evaluate the improvement effects of alpha lipoic acid (ALA), ascorbic acid-6-palmitate (AA6P), fish oil (FO), and their combination (COM) on some biochemical properties in erythrocytes of streptozotocin (STZ)-induced diabetic male rats. According to experimental results, glutathione (GSH) level in erythrocytes decreased in diabetes ($P < 0.01$), D + ALA, and D + AA6P groups ($P < 0.001$). Malonaldehyde (MA) level increased in diabetes ($P < 0.05$), D + FO, and D + COM groups ($P < 0.001$), but its level in D + AA6P and D + ALA groups was lower in diabetes group ($P < 0.01$). Total lipid level in diabetes and diabetes plus antioxidant administered groups were higher than control. Total cholesterol level was high in diabetes and D + ALA groups ($P < 0.05$), but its level reduced in D + FO compared to control and diabetes groups, $P < 0.05$, < 0.001, respectively. Total **triglyceride** (TTG) level was high in the D + ALA ($P < 0.05$) and D + COM ($P < 0.001$) groups. In contrast, TTG level in blood of diabetes group was higher than diabetes plus antioxidant and FO administered groups ($P < 0.001$). According to gas chromatography analysis results, while the palmitic acid raised in diabetes group ($P < 0.05$), stearic acid in D + FO, D + ALA, and diabetes groups was lower than control ($P < 0.05$), **oleic acid** reduced in D + COM and D + FO groups, but its level raised in D + AA6P and D + ALA groups ($P < 0.01$). As the **linoleic acid** (LA) elevated in ALA + D, D + AA6P, and diabetes groups, **linolenic acid** level in diabetes, D + AA6P, and D + FO

groups was lower than control (P < 0.001). Arachidonic acid (AA) decreased in D + ALA, D + AA6P, and diabetes groups (P < 0.01), but its level in D + COM and D + FO was higher than control (P < 0.05). In conclusion, present data have confirmed that the combination of the ALA, AA6P, and FO have improvement effects on the recycling of GSSG to reduced GSH in erythrocytes of diabetic rats, and in addition to this, oxidative stress was suppressed by ALA and AA6P, and unsaturated fatty acid degree was raised by the effects of ALA and FO.

L37 ANSWER 22 OF 56 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:465563 BIOSIS
DOCUMENT NUMBER: PREV200200465563
TITLE: Effect of short-term fatty acid infusion on glucose and **lipid metabolism** in late gestation fetal sheep.
AUTHOR(S): Smith, Danielle L. [Reprint author]; Thureen, Patti J. [Reprint author]; Hay, William W. [Reprint author]
CORPORATE SOURCE: Pediatrics, U of Colorado School of Medicine, Denver, CO, USA
SOURCE: Pediatric Research, (April, 2002) Vol. 51, No. 4 Part 2, pp. 308A. print.
Meeting Info.: Annual Meeting of the Pediatric Societies'. Baltimore, MD, USA. May 04-07, 2002.
CODEN: PEREBL. ISSN: 0031-3998.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Sep 2002
Last Updated on STN: 4 Sep 2002
ED Entered STN: 4 Sep 2002
Last Updated on STN: 4 Sep 2002

L37 ANSWER 23 OF 56 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:91852 BIOSIS
DOCUMENT NUMBER: PREV200100091852
TITLE: Lipid and fatty acid imbalance and neutrophil function in cystic fibrosis.
AUTHOR(S): Nixon, L. S. [Reprint author]; Ionescu, A. A. [Reprint author]; Shale, D. J. [Reprint author]
CORPORATE SOURCE: Section of Respiratory Medicine, Academic Centre, University of Wales College of Medicine, Llandough Hospital, Penarth, Cardiff, CF64 2XX, UK
SOURCE: Thorax, (December, 2000) Vol. 55, No. Supplement 3, pp. A66. print.
Meeting Info.: Winter Meeting of the British Thoracic Society. Westminster, London, UK. December 13-15, 2000. British Thoracic Society.
CODEN: THORA7. ISSN: 0040-6376.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Feb 2001
Last Updated on STN: 12 Feb 2002
ED Entered STN: 21 Feb 2001
Last Updated on STN: 12 Feb 2002

L37 ANSWER 24 OF 56 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 1997:108716 BIOSIS
DOCUMENT NUMBER: PREV199799407919
TITLE: Effect of omega-3 fatty...

CORPORATE SOURCE: Nutr. Metab. Res. Group, Dep. Agric. Food Nutr. Sci., Univ. Alberta, 4-10 Agric./Forestry Cent., Edmonton, AB T6G 2P5, Canada
SOURCE: Diabetologia, (1997) Vol. 40, No. 1, pp. 45-52.
CODEN: DBTGAJ. ISSN: 0012-186X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Mar 1997
Last Updated on STN: 10 Mar 1997
ED Entered STN: 10 Mar 1997
Last Updated on STN: 10 Mar 1997
AB This study was conducted to examine the effect of **omega-3 fatty acid** supplementation on plasma lipid, cholesterol and lipoprotein fatty acid content of non-insulin-dependent diabetic individuals consuming a higher (0.65, n = 10) or lower (0.44, n = 18) ratio of dietary polyunsaturated to saturated fatty acid (P/ S). The participants were initially given an **olive oil** supplement (placebo) equivalent to 35 mg of 18:1 cntdot kg body weight-1 cntdot day-1 for 3 months. This was followed by two omega-3 supplement periods in a randomized crossover. In these 3-month periods, participants were given a **linseed oil** supplement equivalent to 35 mg of 18:3-omega-3 cntdot kg body weight-1 cntdot day-1 or a fish oil supplement equivalent to 35 mg of 20:5-omega-3 + 22:6-omega-3 cntdot kg body weight-1 cntdot day-1. At the end of each supplement period, a blood sample was drawn from each participant for lipid, lipoprotein, insulin, glucagon and C-peptide analyses. At the end of each 3-month period a 7-day dietary record was completed to calculate dietary fat intake and P/S ratio. Results indicate that fish oil significantly reduced plasma **triacylglycerol** level (p lt 0.05) and increased 20:5-omega-3 and 22:6-omega-3 content of all lipoprotein lipid classes. **Linolenic acid** supplementation had no effect on plasma **triacylglycerol** level, but it increased 18:3-omega-3 content of lipoprotein cholesterol ester fractions (p lt 0.05). A slight increase in 20:5-omega-3, but not 22:6-omega-3, content was noted in lipoprotein lipid classes as a result of 18:3-omega-3 supplementation. LDL and HDL cholesterol, insulin, glucagon and C-peptide levels were not affected by either omega-3 supplement. It is concluded that a modest intake of **omega-3 fatty acids**, such as could be obtained from consuming fish regularly, will reduce plasma **triglyceride** level without affecting LDL or HDL cholesterol levels.

L37 ANSWER 25 OF 56 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1981:241413 BIOSIS
DOCUMENT NUMBER: PREV198172026397; BA72:26397
TITLE: INFLUENCE OF PANTETHINE ON PLATELET VOLUME MICRO VISCOSITY LIPID COMPOSITION AND FUNCTIONS IN **DIABETES MELLITUS** WITH HYPER LIPIDEMIA.
AUTHOR(S): HIRAMATSU K [Reprint author]; NOZAKI H; ARIMORI S
CORPORATE SOURCE: FOURTH DEP INTERN MED, SCH MED, TOKAI UNIV, BOHSEIDAI, ISEHARA, KANAGAWA 259-11, JPN
SOURCE: Tokai Journal of Experimental and Clinical Medicine, (1981) Vol. 6, No. 1, pp. 49-58.
CODEN: TJEMDR. ISSN: 0385-0005.
DOCUMENT TYPE: Article
FILE SEGMENT: BA

LANGUAGE: ENGLISH

AB The in vivo effects of pantethine were investigated on serum lipids and platelet lipid and platelet functions in 31 diabetic patients with healthy platelets. The characteristics of lipid composition in diabetic platelets were high levels of free cholesterol, phospholipid, **triglyceride**, cholesterol ester, palmitoleic acid, linolic acid and palmitoleic acid/palmitic acid and low levels of the molar ratio of free cholesterol/phospholipid, phosphatidylethanolamine, **oleic acid**, arachidonic acid and **oleic acid**/stearic acid. Pantethine normalized these values of fatty acids to the control levels, and concomitantly reduced significantly the hyperaggregation of platelets induced by 106 M ADP and the hyper-ADP release reaction from platelets when exposed to 2 μg collagen, and made the volume smaller and the microviscosity lower after oral administration. Pantethine apparently normalized the abnormalities of serum lipids and platelet lipid compositions and subsequently reduced the hyperaggregation and hyperrelease reactions through the changes of volume and microviscosity of the platelets in **diabetes mellitus** with hyperlipidemia.

L37 ANSWER 26 OF 56 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 1978:224033 BIOSIS
DOCUMENT NUMBER: PREV197866036530; BA66:36530
TITLE: EFFECT OF GLUCOSE OR OIL SUPPLEMENTATION ON LIPOGENIC ENZYMES IN OVER FED CHICKS.
AUTHOR(S): SHAPIRA N [Reprint author]; NIR I; BUDOWSKI P
CORPORATE SOURCE: DEP ANIM SCI, FAC AGRIC, HEB UNIV JERUS, REHOVOT, ISR
SOURCE: Journal of Nutrition, (1978) Vol. 108, No. 3, pp. 490-496. CODEN: JONUAI. ISSN: 0022-3166.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB Chicks of a light breed [New Hampshire + Leghorn], aged 26 days, were force-fed by intubation for 10 days. The feed administered consisted of a basal low-fat closed formula cereal based diet and of supplements of either glucose or soybean oil supplied in isoenergetic amounts. Ad lib-fed chicks served as controls. At the end of the experiment, the weights and lipid contents of carcass, livers and abdominal adipose tissue were determined, as was the glucose and lipid content of plasma. The activities of citrate cleavage and malic enzymes were assayed in livers and adipose tissue. The increased weight and lipid content caused by glucose or oil supplements were similar in carcass and adipose tissue, but liver weight and fat content were increased much more by the glucose supplement than by the oil. The lipids which accumulated in the livers of force-fed chicks were essentially **triglycerides**. The glucose supplement caused a pronounced drop in the **linoleic acid** content of liver lipids, as well as an increase in **oleic acid**; the oil produced the opposite effect. The glucose supplement caused a more pronounced **hyperglycemia** and hyperlipemia than did the oil. Activities of citrate cleavage and malic enzymes increased in proportion to the amount of carbohydrates force-fed in excess of the control intake. These and previous results show that chicks adapt to excess carbohydrate intake by both liver enlargement and increased activity of lipogenic enzymes.

L37 ANSWER 27 OF 56 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2004:80358 HCAPLUS
DOCUMENT NUMBER: 140:139513
TITLE: Methods of identifying compounds that affect a fatty

acid cell-surface receptor
INVENTOR(S): Owman, Christer; Olde, Bjorn; Kotarsky, Knut; Nilsson, Niclas; Fladgren, Erik

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004019109	A1	20040129	US 2002-202687	20020724

PRIORITY APPLN. INFO.: US 2002-202687 20020724
ED Entered STN: 01 Feb 2004
AB The present invention provides methods for screening and identifying compds. that affect the **metab**. of fatty acids and fatty acid derivs., and thus for compds. that possess anti-diabetic as well as anti-obesity properties and possess the ability to affect the levels of chylomicrons, **triacylglycerols**, cholesterols, and fatty acids in a patient. Kits and compns. for screening and identifying such compds. are also provided. The invention is predicated on the identification of a physiol. receptor for free fatty acids and anti-diabetic and anti-obesity drugs.

L37 ANSWER 28 OF 56 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:612070 HCAPLUS
DOCUMENT NUMBER: 141:134104
TITLE: **Lipid metabolism**-improving and insulin resistance-decreasing agents containing diglycerides an foods containing them for diabetic patients
INVENTOR(S): Mizuno, Tomohito; Watanabe, Hiroyuki
PATENT ASSIGNEE(S): Kao Corp., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 15 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2004210652	A2	20040729	JP 2002-379276	20021227

PRIORITY APPLN. INFO.: JP 2002-379276 20021227
ED Entered STN: 30 Jul 2004
AB The **lipid metab**.-improving and insulin resistance-decreasing agents for diabetic patients, contain diglycerides showing .**omega**.-**3 fatty acid** content (in constituent fatty acids) of <15 weight%. The agents decrease **triglycerides** in blood lipoproteins (e.g., LDL) of diabetic patients and increase cholesterol in blood HDL. Fatty acids were obtained by hydrolysis of **rape oil**, subjected to wintering for lowering of saturated fatty acid content, and esterified with glycerin in the presence of Lipozyme 3A (1,3-selective lipase) at 40° to give an oil composition showing a glyceride composition of **triglycerides** 12.98, diglycerides 85.89, and monoglycerides 1.06% and a constituent fatty acid composition of C16 3.16, C18 1.27, C18:1 37.49, C18:2 48.27, and C18:3 6.36%. Patients with **type 2 diabetes mellitus** were divided into 2 groups and given .apprx.10 g/day of the composition (as a substitute for normal oil) (test group) or a control oil composition (**triglyceride** content 97.78%) (control group) for 3 mo. Serum LDL **triglyceride** concns. of the test group and control

group were 84.1 and 111.4%, resp., and serum HDL cholesterol concns. of these 2 groups were 116.6 and 112.6%, resp., after the 3-mo test period (these concns. at 0 mo were considered 100%).

TITLE: Associations between the fatty acid content of **triglyceride**, visceral adipose tissue accumulation, and components of the insulin resistance syndrome

AUTHOR(S): Tremblay, Andre J.; Despres, Jean-Pierre; Piche, Marie-Eve; Nadeau, Andre; Bergeron, Jean; Almeras, Natalie; Tremblay, Angelo; Lemieux, Simone

CORPORATE SOURCE: Department of Food Sciences and Nutrition, Laval University, Ste-Foy, Quebec, Can.

SOURCE: Metabolism, Clinical and Experimental (2004), 53(3), 310-317
CODEN: METAAJ; ISSN: 0026-0495

PUBLISHER: Elsevier Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 25 Feb 2004

AB Many factors are involved in the development of the insulin resistance syndrome, such as visceral obesity and the type of dietary fat. The main purpose of this study was to investigate the relationships between fatty acid content of **triglyceride** (TG), visceral adipose tissue (AT) accumulation, and **metabolic** components of the insulin resistance syndrome in a group of 97 Caucasian men with a mean age of 45.1±7.2 yr (29 to 63 yr). To reach these objectives, Spearman correlations, group comparisons, and stepwise multiple regression analyses were performed. The proportion of palmitic acid (16:0) in the TG fraction was pos. associated with plasma fasting insulin (r =.25, P =.03), diastolic (r =.45, P <.001), and systolic (r =.29, P =.003) blood pressure. On the other hand, the proportion of **.alpha.-linolenic acid** (18:3n-3) was associated neg. with apolipoprotein (apo) B (r = -.29, P =.005) and pos. with low-d. lipoprotein (LDL) diameter (r =.29, P =.007), while the proportion of **γ- linolenic acid** (18:3n-6) was associated neg. with plasma TG (r = -.33, P =.003), diastolic (r = -.29, P =.01), and systolic (r = -.35, P =.002) blood pressure and plasma fasting insulin (r = -.37, P =.0005) and pos. with high-d. lipoprotein (HDL)2-cholesterol (r =.27, P =.01) and LDL diameter (r =.25, P =.02). Stepwise multiple regression analyses were conducted to determine the contribution of visceral AT, body fat mass, and the fatty acid content of TG to the variance of **metabolic** variables studied. It was found that visceral AT contributed significantly to the variance in plasma TG (R2 = 20.7%, P <.0001), apo B (R2 = 9.0%, P =.007), HDL2-cholesterol (R2 = 17.9%, P <.0001), LDL diameter (R2 = 4.9%, P =.02), and area under the glucose curve (AUC-glucose) (R2 = 8.2%, P =.006). On the other hand, body fat mass contributed significantly to the variance in fasting insulin (R2 = 19.7%, P <.0001) and diastolic (R2 = 6.8%, P =.007) and systolic (R2 = 10.5%, P =.01) blood pressure. At least one fatty acid made a significant contribution to the variance of each **metabolic** variable studied. In fact, the proportion of 18:3n-6 contributed significantly to the variance in both TG (R2 = 8.9%, P = 0.007) and HDL2-cholesterol (R2 = 6.0%, P =.01). Moreover, 18:3n-3 contributed to the variance of apo B (R2 = 7.0%, P =.02), while 18:3n-6 made the largest contribution to the variance of LDL diameter (R2 = 7.6%, P =.02). Finally, 16:0 significantly contributed to the variance of AUC-glucose (R2 = 11.4%, P =.0003), diastolic (R2 = 25.2%, P <.0001), and systolic (R2 = 6.8%, P =.002) blood pressure. In summary, results of this study suggest that the fatty acid content of TG is associated with many **metabolic** variables of the insulin resistance syndrome independently of body fat mass or visceral AT accumulation.

REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

in the pathogenesis of diabetic cardiomyopathy: Modulation by dietary fat content
AUTHOR(S): Finck, Brian N.; Han, Xianlin; Courtois, Michael; Aimond, Franck; Nerbonne, Jeanne M.; Kovacs, Attila; Gross, Richard W.; Kelly, Daniel P.
CORPORATE SOURCE: Department of Medicine, Center for Cardiovascular Research, Washington University School of Medicine, St. Louis, MO, 63110, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2003), 100(3), 1226-1231
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 16 Feb 2003
AB To explore the role of peroxisome proliferator-activated receptor α (PPARα)-mediated derangements in myocardial **metab.** in the pathogenesis of diabetic cardiomyopathy, insulinopenic mice with PPARα deficiency (PPARα-/-) or cardiac-restricted overexpression [myosin heavy chain (MHC)-PPAR] were characterized. Whereas PPARα-/- mice were protected from the development of diabetes-induced cardiac hypertrophy, the combination of diabetes and the MHC-PPAR genotype resulted in a more severe cardiomyopathic phenotype than either did alone. Cardiomyopathy in diabetic MHC-PPAR mice was accompanied by myocardial long-chain **triglyceride** accumulation. The cardiomyopathic phenotype was exacerbated in MHC-PPAR mice fed a diet enriched in **triglyceride** containing long-chain fatty acid, an effect that was reversed by discontinuing the high-fat diet and absent in mice given a **medium-chain triglyceride**-enriched diet. Reactive oxygen intermediates were identified as candidate mediators of cardiomyopathic effects in MHC-PPAR mice. These results link dysregulation of the PPARα gene regulatory pathway to cardiac dysfunction in the diabetic and provide a rationale for serum lipid-lowering strategies in the treatment of diabetic cardiomyopathy.
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L37 ANSWER 31 OF 56 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:83842 HCAPLUS
DOCUMENT NUMBER: 139:63044
TITLE: A human cell surface receptor activated by free fatty acids and thiazolidinedione drugs
AUTHOR(S): Kotarsky, Knut; Nilsson, Niclas E.; Flodgren, Erik; Owman, Christer; Olde, Bjorn
CORPORATE SOURCE: Division of Molecular Neurobiology, Wallenberg Neuroscience Center, Lund, SE-221 84, Swed.
SOURCE: Biochemical and Biophysical Research Communications (2003), 301(2), 406-410
CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER: Elsevier Science
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 03 Feb 2003
AB Fatty acids, which are essential nutritional components, are also involved in cardiovascular and **metabolic** diseases. Here we report a human cell surface receptor that we name free fatty acid receptor (FFAR), because it is specifically activated by medium to long-chain free fatty

acids. The receptor belongs to the class of seven-transmembrane, G-protein coupled receptors (GPCRs) and also mediates responses to antidiabetic drugs of the thiazolidinedione type. It is expressed in

form. In view of the nature of the activating substances, their physiol. role in the body, and the tissue distribution of FFAR we suggest the term "nutrient sensing receptor" for receptors acting at the interface between dietary components and signaling mols.

REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L37 ANSWER 32 OF 56 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2000:882249 HCAPLUS
DOCUMENT NUMBER: 134:176714
TITLE: Diabetes-induced changes in specific lipid molecular species in rat myocardium
AUTHOR(S): Han, Xianlin; Abendschein, Dana R.; Kelley, John G.; Gross, Richard W.
CORPORATE SOURCE: Division of Bioorganic Chemistry and Molecular Pharmacology, Washington University School of Medicine, St. Louis, MO, 63110, USA
SOURCE: Biochemical Journal (2000), 352(1), 79-89
CODEN: BIJOAK; ISSN: 0264-6021
PUBLISHER: Portland Press Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 17 Dec 2000
AB Intrinsic cardiac dysfunction during the diabetic state has been causally linked to changes in myocardial **lipid metab.** However, the precise alterations in the mol. species of myocardial polar and non-polar lipids during the diabetic state and their responses to insulin have not been investigated. Herein we demonstrate four specific alterations in rat myocardial lipid mol. species after induction of the diabetic state by streptozotocin treatment: (i) a massive remodelling of **triacylglycerol** mol. species including a > 5-fold increase in tripalmitin mass and a 60% decrease in polyunsatd. **triacylglycerol** mol. species mass (i.e. **triacylglycerols** containing at least one acyl residue with more than two double bonds); (i) a 46% increase in myocardial phosphatidylinositol mass; (iii) a 44% increase in myocardial plasmenylethanolamine mass and (iv) a 22% decrease in 1-stearoyl-2-arachidonoyl phosphatidylethanolamine content. Each of the changes in phospholipid classes, subclasses and individual mol. species were prevented by insulin treatment after induction of the diabetic state. In sharp contrast, the alterations in **triacylglycerol** mol. species were not preventable by peripheral insulin treatment after induction of the diabetic state. These results segregate diabetes-induced alterations in myocardial **lipid metab.** into changes that can be remedied or not by routine peripheral insulin treatment and suggest that peripheral insulin therapy alone may not be sufficient to correct all of the **metabolic** alterations present in diabetic myocardium.
REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L37 ANSWER 33 OF 56 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1999:644013 HCAPLUS
DOCUMENT NUMBER: 131:321930
TITLE: Palmitate and **oleate** induce the immediate-early response genes c-fos and nur-77 in the pancreatic β-cell line INS-1
AUTHOR(S): Roche, Enrique; Buteau, Jean; Aniento, Inmaculada; Reig, Juan Antonio; Soria, Bernat; Prentki, Marc

CORPORATE SOURCE: Instituto de Bioingenieria/Division of Nutrition
University Miguel Hernandez, Alicante, Spain
SOURCE: Diabetes (1999), 48(10), 2007-2014

ED Entered STN: 11 Oct 1999
AB To better understand the link between fatty acid signaling and the pleiotropic effects of fatty acids in the pancreatic β-cells, we investigated whether fatty acids regulate immediate-early response genes (IEGs) coding for transcription factors implicated in cell proliferation, differentiation, and apoptosis. Palmitate and **oleate**, but not long-chain polyunsatd. fatty acids, caused a pronounced accumulation of c-fos and nur-77 mRNAs in β-cells (INS-1 cells) to an extent similar to that produced by the protein kinase C (PKC) activator phorbol myristate acetate (PMA). The effect was dose-dependent and occurred at concns. 0.1-0.5 mM in the presence of 0.5% albumin. The action of the 2 fatty acid occurred at the transcriptional level and the mRNA accumulation displayed a bell-shaped kinetics with a maximal effect at 1 h. Since 2-bromopalmitate was ineffective, the fatty acids must be **metabolized** first to induce this effect. Neither fatty acid induced c-fos and nur-77 in PKC-downregulated cells or cells incubated in the presence of the Ca2+ channel blocker nifedipine or the Ca2+ chelator EGTA, suggesting an involvement of the PKC and Ca2+ signaling pathways. Palmitate and **oleate** also increased the c-fos protein expression and DNA binding activity of the transcription factor AP-1. **Oleate** , but not palmitate, increased the [3H]thymidine incorporation in INS cells. Both palmitate and **oleate** caused c-fos and nur-77 mRNA accumulation in isolated rat islets. The IEG induction by the most abundant circulating fatty acids may play a role in the adaptive processes of pancreatic β-cells to hyperlipidemia. The results have implications for the understanding of obesity-associated **diabetes mellitus** and the link between fatty acids and tumorigenesis.
REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L37 ANSWER 34 OF 56 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1998:274251 HCAPLUS
DOCUMENT NUMBER: 129:15559
TITLE: Effect of selenium and vitamin E supplements on tissue lipids, peroxides, and fatty acid distribution in experimental diabetes
AUTHOR(S): Douillet, C.; Bost, M.; Accominotti, M.; Borson-Chazot, F.; Ciavatti, M.
CORPORATE SOURCE: National Institute of Health and Medical Research Unit 331, Bron, 69375, Fr.
SOURCE: Lipids (1998), 33(4), 393-399
CODEN: LPDSAP; ISSN: 0024-4201
PUBLISHER: AOCS Press
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 13 May 1998
AB The effects of dietary Se given in Se-rich yeasts, selenomethionine, or selenomethionine + vitamin E supplements on lipids, lipid peroxides, and fatty acid distribution in body tissues were studied in 76 male Sprague-Dawley rats with streptozotocin-induced **diabetes mellitus** after 24 wk of disease. Diabetes increased the liver thiobarbituric acid-reactive substances and conjugated dienes; Se supplement completely corrected these changes. In the kidneys and heart, the peroxide levels were not changed by diabetes. In the liver, a significant drop in **triglycerides** and phospholipids was observed; this was modulated by Se + vitamin E supplementation. Se + vitamin E also inhibited the decrease in 18:2n-6 and the increase in 22:6n-3 fatty acids

observed in the liver of diabetic rats; these changes reflected the altered glycemic control. In the kidney, heart and aorta, **diabetes**

increased the level of 20:5n-3 fatty acid. These polyunsatd. fatty acids are precursors of PGI2 and PGI3 which may protect against cardiovascular dysfunctions. In the kidney, Se decreased the levels of 20:4n-6 fatty acid, the precursor of TXA2 implicated in the diabetic glomerular injury. Thus Se, and more efficiently Se + vitamin E supplementation in exptl. **diabetes mellitus**, could play a role in controlling the oxidative status and altered **lipid metab**. in the liver, thereby maintaining favorable fatty acid distribution in the major tissues affected by diabetic complications.

REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L37 ANSWER 35 OF 56 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1998:767573 HCAPLUS
DOCUMENT NUMBER: 130:109581
TITLE: Effect of enteral nutritional products differing in carbohydrate and fat on indices of carbohydrate and **lipid metabolism** in patients with NIDDM
AUTHOR(S): McCargar, Linda J.; Innis, Sheila M.; Bowron, Elaine; Leichter, Joseph; Dawson, Keith; Toth, Ellen; Wall, Katherine
CORPORATE SOURCE: Department of Agricultural Food & Nutritional Science, University of Alberta, Edmonton, T6G 2P5, Can.
SOURCE: Molecular and Cellular Biochemistry (1998), 188(1&2), 81-89
CODEN: MCBIB8; ISSN: 0300-8177
PUBLISHER: Kluwer Academic Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 08 Dec 1998
AB Non-insulin dependent **diabetes mellitus** (NIDDM) is associated with chronic **hyperglycemia**, which increases the risk of vascular complications. Elevated **triglyceride** (TG) and VLDL cholesterol levels and low levels of HDL cholesterol have been frequently found in NIDDM patients. Diets high in complex carbohydrates and low in fats are typically recommended for NIDDM management, but this has recently been challenged by reports of benefits from diets high in monounsatd. fat. The effects of diets high in fiber (Ensure with Fiber, 30% fat) or in **monounsatd. fatty acids** (Glucerna, 50% fat) were studied in 32 NIDDM individuals. The products were consumed for 28 days at >80% of daily energy intake. The dietary compliance was verified by higher blood plasma glyceride **oleic acid** levels in the Glucerna group and higher plasma glyceride **linoleic acid** levels in the Ensure with Fiber group. The postprandial rise in blood glucose levels was lower in the Glucerna group. The trends of clin. interest included greater average decreases in the Glucerna group compared with the Ensure/Fiber group in the levels fructosamine (by 9.13 vs. 0.14 μM), glucose (by 1.61 vs. 0.63 mM), and insulin (by 46.0 vs. 12.6 pM) during the study. However, the fasting plasma glucose, fructosamine, glyceride, and cholesterol levels were not different between groups. Thus, the high-monounsatd. fat diet and the standard diet were similar with regard to usual indicators of carbohydrate and **lipid metab**. High-monounsatd. fat diets appears to pose no risk to lipoprotein **metab**. in NIDDM patients.

REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L37 ANSWER 36 OF 56 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1990:53194 HCAPLUS

Valerie; Neville, M. C.; Seacat, Joy; Wood, D. L.
CORPORATE SOURCE: Milk Secret. Mastitis Lab., U S Dep. Agric., Beltsville, MD, 20705, USA
SOURCE: American Journal of Clinical Nutrition (1989), 50(6), 1364-9
CODEN: AJCNAC; ISSN: 0002-9165
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 17 Feb 1990
AB Milk volume and composition were examined in a diabetic mother on days 3-7 postpartum. By day 5 milk volume produced and concns. of Na, K, chloride, lactose, protein, Ca, Mg, and citrate were within limits of a reference population. Fat content of the milk was slightly lower. Free fatty acids were 2% of total lipid on day 3 but increased to 23% on days 4-7, suggesting impaired esterification in the mammary gland. Total milk lipoprotein lipase increased .apprx.4-fold during days 4-5. Other changes were (1) low cholesterol content, only 1/5th of normal milk; (2) decreased medium-chain fatty acids, suggesting impairment of fatty acid synthesis in the mammary gland; (3) increased **oleic acid**; and (4) high concns. of polyunsatd. fatty acids, suggesting increased chain elongation. Apparently, diabetes produces changes in **lipid metab.** in the mammary gland that alter the composition of milk produced by the diabetic mother.

L37 ANSWER 37 OF 56 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1989:191602 HCAPLUS
DOCUMENT NUMBER: 110:191602
TITLE: **Linoleic-acid**-enriched diet: long-term effects on serum lipoprotein and apolipoprotein concentrations and insulin sensitivity in noninsulin-dependent diabetic patients
AUTHOR(S): Heine, Robert J.; Mulder, Cees; Popp-Snijders, Corrie; Van der Meer, Jan; Van der Veen, Ed A.
CORPORATE SOURCE: Dep. Intern. Med., Free Univ. Hosp., Amsterdam, Neth.
SOURCE: American Journal of Clinical Nutrition (1989), 49(3), 448-56
CODEN: AJCNAC; ISSN: 0002-9165
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 26 May 1989
AB Long-term (30 wk) effects on serum lipoproteins and insulin sensitivity of 2 diets, one with a low polyunsatd.-to-saturated fat ratio (P:S 0.3) and one with a P:S of 1.0, were compared in patients with noninsulin-dependent **diabetes mellitus** (NIDDM) in a crossover study. Total and low-d. lipoprotein-cholesterol levels declined by 7.6% and 9.8%, resp., during the high P:S diet. Very-low-d. lipoprotein-, high-d. lipoprotein-2-, and high-d. lipoprotein-3-cholesterol; **triacylglycerol**; and apolipoprotein A1, A2, and B levels were not affected by the change in P:S. Despite a modest increase of insulin-mediated glucose disposal at physiol. insulinemia during the high P:S diet, no influence was seen on glycemic control and on blood glucose, plasma insulin, and C peptide responses to mixed meals. In conclusion, a **linoleic acid**-enriched diet in patients with NIDDM causes a less atherogenic lipoprotein profile but does not influence glycemic control and carbohydrate tolerance.

L37 ANSWER 38 OF 56 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1988:608732 HCAPLUS
DOCUMENT NUMBER: 109:208732
TITLE: Studies on the optimized requirement of daily protein

SOURCE: Tairyoku Kenkyu (1988), 69, 167-74
CODEN: TAKNAS; ISSN: 0389-9071
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
ED Entered STN: 10 Dec 1988
AB By use of rat models, basic features of the enzyme of the 1st step in the pathway leading to arginine formation, pyrroline-5-carboxylate synthase, were studied. A highly sensitive and specific assay procedure for the enzyme was developed. This enabled detection of lower activities in a number of tissues such as thymus, pancreas, and lymph node. After an extensive survey of as many as 30 tissues, it was found that this enzyme was essentially localized to the upper small intestine. These was no difference in the activity between male and female rats. Some analgesics, such as aspirin and aminopyrine, and some **metabolites** of tyrosine, such as gentisic acid and dopamine, inhibited the activity. Exptl. diabetes and aging both caused a decrease in pyrroline-5-carboxylate synthase activity in the small intestine. The importance of the activity in man for optimized physiol. is discussed.

L37 ANSWER 39 OF 56 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1984:100496 HCAPLUS
DOCUMENT NUMBER: 100:100496
TITLE: Role of membrane phospholipids and fatty acids in the mechanism of regulating glucose utilization by tissues
AUTHOR(S): Isaev, E. I.; Ergashova, M. Zh.; Bornikov, V. T.; Nikolaeva, N. F.; Saatov, T. S.; Turakulov, Ya. Kh.
CORPORATE SOURCE: Inst. Biokhim., Tashkent, USSR
SOURCE: Uzbekskii Biologicheskii Zhurnal (1958-199?) (1983), (5), 3-5
CODEN: UZBZAZ; ISSN: 0042-1685
DOCUMENT TYPE: Journal
LANGUAGE: Russian
ED Entered STN: 12 May 1984
AB Glucose uptake by rat cardiac, skeletal muscle, and hepatic tissue was altered in the presence of phospholipids and fatty acids. Phosphatidic, butyric, and **caprylic acids** increased glucose uptake in all tissues by up to 2-fold. The effect of the fatty acids was dependent on their degree of unsatn.; thus, **linoleic acid** was more active than **oleic acid**. In expts. in vivo, insulin increased levels of unsatd. fatty acids in the above tissues; during **hyperglycemia**, saturated fatty acid levels increased.

L37 ANSWER 40 OF 56 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1977:515856 HCAPLUS
DOCUMENT NUMBER: 87:115856
TITLE: Gas chromatographic studies on the composition of the fatty acid spectrum of the **triglycerides** and cholesterol esters in diabetics and people with healthy **metabolism**
AUTHOR(S): Gnauck, G.; Singer, P.; Honigmann, G.
CORPORATE SOURCE: Fed. Rep. Ger.
SOURCE: Diabetes Epidemiol. Eur. (1975), 112-15. Editor(s): Gutsche, Horst; Holler, Heinz D. Thieme: Stuttgart, Ger.
CODEN: 36EBA6
DOCUMENT TYPE: Conference

LANGUAGE: English
ED Entered STN: 12 May 1984
AB After administering a standard diet, in diabetics there was a fall in

between healthy subjects and diabetics were slight. If, however, there was >10% excess weight, then the ratio of **oleic acid** to **linoleic acid** of esterified cholesterol in the nondiabetic was changed. In people with normal weight, the ratio was .apprx.1:2, and in obese subjects it reached a ratio of 2:1.

L37 ANSWER 41 OF 56 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1967:498559 HCAPLUS
DOCUMENT NUMBER: 67:98559
TITLE: Selective mobilization of fatty acids from the adipose tissue of obese **hyperglycemic** mice
AUTHOR(S): Stein, Janet; Anderson, John; Hollifield, Guy
CORPORATE SOURCE: Univ. of Virginia Med. Sch., Charlottesville, VA, USA
SOURCE: Metabolism, Clinical and Experimental (1967), 16(8), 758-62
CODEN: METAAJ; ISSN: 0026-0495
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 12 May 1984
AB After starvation of normal mice the ovarian fat pad **triglycerides** showed an increase in the proportion of **oleic acid** and no change in the proportion of **linoleic acid**. Starved obese **hyperglycemic** mice on the other hand, showed no change in **oleic acid** and an increase in **linoleic acid**. Also, monoglyceride lipase activity was considerably lower in the adipose tissue of the obese mice. Linoleic and **oleic acids** together make up 88% of the fatty acids in the 2-position of the **triglyceride** of fat samples of fed obese mice, yet only the proportion of **linoleic acid** increased during starvation of these mice. Since **linoleic acid** is an essential fatty acid and cannot be synthesized, its increased proportion must be explained by its preferential retention. The following hypothesis is offered to explain the findings: the lower rate of activity of monoglyceride lipase in the fat of starved obese mice leads to the selective intracellular retention of **linoleic acid** as the 2-monoglyceride, which may then be recycled back to **triglyceride** by direct acylation at the 1- and 3-positions.

L37 ANSWER 42 OF 56 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2005121348 EMBASE
TITLE: Oleoyl-estrone **metabolic** effects in relation with caloric restriction in inbred Beta rats with spontaneous obesisty and **type 2 diabetes**.
AUTHOR: Posadas M.D.; Olguin M.C.; Zingale M.I.; Revelant G.; Labourdette V.; Gayol M.D.C.; Calderari S.
CORPORATE SOURCE: Dr. M.C. Olguin, Catedra de Bromatologia, Fac. Cie. Bioquimicas/Farmaceuticas, Suipacha 531, 2000 Rosario, Argentina. molguin@fbioyf.unr.edu.ar
SOURCE: Medicina, (2004) Vol. 64, No. 4, pp. 332-336.
Refs: 21
ISSN: 0025-7680 CODEN: MEDCAD
COUNTRY: Argentina
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
005 General Pathology and Pathological Anatomy
029 Clinical Biochemistry

030 Pharmacology
037 Drug Literature Index
LANGUAGE: English

Last Updated on STN: 20050407
AB Spontaneously hypertriacylglycerolemic obese and diabetic inbred IIM Beta rats were treated with oleoyl-estrone for 10 days. Pair-feeding was performed to determine some oleoyl-estrone effects dependent on the caloric restriction it promotes. Twenty-five 200 day-old Beta males receiving a daily gavage of 0.2 ml **sunflower oil** were divided into the following groups: 1) daily dose of 10 nmol/g oleoyl-estrone; 2) pair-fed; 3) control. The variables measured were: whole body protein, water and lipid; retroperitoneal and epididymal fat depot weights; plasma urea, glucose, insulin, **triacylglycerols** and cholesterol. Biomass and food intake were assessed daily. Oleoyl-estrone and pair-fed groups expressed similar variations in body composition and significant body weight losses due to reduction in food intake. Oleoyl-estrone and pair-fed treatments significantly reduced retroperitoneal fat depot weights, but not epididymal ones. In oleoyl-estrone and pair-fed groups **hyperglycemia** decreased and insulinemia lowered significantly. Plasma normal total cholesterolemia and hypertriacylglycerolemia values typical of Beta rats decreased strongly compared to controls, though attaining significantly different values between oleoyl-estrone and pair-fed groups. Plasma total cholesterol appeared as more sensitive to caloric restriction than **triacylglycerols** through a specific oleoyl-estrone-mediated effect.

L37 ANSWER 43 OF 56 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2003347426 EMBASE
TITLE: Influence of plasma free fatty acids on lipoprotein synthesis and diabetic dyslipidemia.
AUTHOR: Julius U.
CORPORATE SOURCE: Dr. U. Julius, Med. Klinik und Poliklinik III, Univ. Klin. Carl Gustav Carus, Fetscherstrasse 74, 01307 Dresden, Germany. julius@rcs.urz.tu-dresden.de
SOURCE: Experimental and Clinical Endocrinology and Diabetes, (2003) Vol. 111, No. 5, pp. 246-250.
Refs: 30
ISSN: 0947-7349 CODEN: ECEDFQ
COUNTRY: Germany
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 003 Endocrinology
005 General Pathology and Pathological Anatomy
006 Internal Medicine
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20030911
Last Updated on STN: 20030911
ED Entered STN: 20030911
Last Updated on STN: 20030911
AB The regulation of hepatic VLDL secretion mainly depends on apolipoprotein (apo) B synthesis, on microsomal **triglyceride** transfer protein, insulin and the availability of **triglycerides**, free fatty acids (FFA) and cholesteryl ester. Four sources of fatty acids are used for lipoprotein synthesis: de-novo lipogenesis, cytoplasmic **triglyceride** stores, fatty acids derived from lipoproteins taken up directly by the liver and plasma FFA. Quantitatively, de-novo lipogenesis plays a minor role in regulating VLDL synthesis, but evidently

it is elevated under conditions of high carbohydrate feeding. Cytoplasmic **triglyceride** stores appear to essentially contribute to VLDL triglycerides. Plasma FFA enter the hepatocytes and are either

shown to stimulate production of VLDL **triglyceride** and apoB. In human beings, an acute experimental elevation of plasma FFA stimulates VLDL production. In healthy men strong positive relations were found between the late increases in large **triglyceride**-rich lipoproteins and plasma FFA concentrations after 6 h following a mixed meal. In contrast, n-3 fatty acids impair VLDL assembly and secretion. Chronic hyperinsulinemia seems to stimulate VLDL production. On the other hand, the short-term addition of insulin has been shown to inhibit VLDL-**triglyceride** and apoB production in vitro. There is in vivo evidence that acute hyperinsulinemia suppresses VLDL-apoB and VLDL-**triglyceride** production in insulin-sensitive humans. Part of this action is due to suppression of plasma FFA. In patients with **impaired glucose tolerance** (IGT), VLDL production was increased when compared with subjects with normal glucose (NGT). When infusing a lipid emulsion, VLDL production could not be further stimulated in IGT patients in contrast to NGT persons. Hypertriglyceridemia in **type 2 diabetes mellitus** is usually the consequence of a VLDL overproduction. In type 2 diabetic patients, in contrast to normal men, insulin failed to suppress VLDL1 particle release. In normal men, an elevation of blood glucose led to a decrease in fatty acid oxidation and an increase in hepatic **triglyceride** secretion. Under these conditions, -30% of total VLDL **triglycerides** coming out of the liver did not originate from plasma FFA. In conclusion, plasma FFA seem to play an important role in stimulating hepatic VLDL production. Other factors such as chronic hyperinsulinemia or nutrition modify this effect.

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ACCESSION NUMBER: 2003347425 EMBASE
TITLE: Treatment of diabetic dyslipoproteinemia.
AUTHOR: Steinmetz A.
CORPORATE SOURCE: A. Steinmetz, St. Nikolaus-Stiftshospital GmbH A., Teaching Hospital, University of Bonn, Hindenburgwall 1, 56626 Andernach, Germany. armin.steinmetz@stiftshospital-andernach.de
SOURCE: Experimental and Clinical Endocrinology and Diabetes, (2003) Vol. 111, No. 5, pp. 239-245.
Refs: 65
ISSN: 0947-7349 CODEN: ECEDFQ
COUNTRY: Germany
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 006 Internal Medicine
018 Cardiovascular Diseases and Cardiovascular Surgery
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20030911
Last Updated on STN: 20030911
ED Entered STN: 20030911
Last Updated on STN: 20030911

AB **Diabetes mellitus**, specifically type 2, is often associated with disorders in **lipid metabolism**. Elevated levels of plasma free fatty acids play a pivotal role by contributing significantly to insulin resistance. In addition free fatty

acids promote diabetic dyslipidemia through increasing VLDL synthesis in the liver, and by virtue of cholesterylester transfer protein, modifying LDL to increase small dense LDL subfractions and to decrease HDL

events, the most common cause of death in **type 2 diabetes**. To decrease the risk of cardiovascular disease events in diabetics, dyslipidemia needs to be treated, as evidenced from epidemiology, from intervention trials, and from subgroup analyses of large intervention trials initiated to evaluate effects of lipid lowering treatment that also included patients with **type 2 diabetes**. Most measures used to counteract **hyperglycemia**, are also prone to ameliorate dyslipidemia: dietary intervention (medical nutrition) including **.omega.-3 fatty acids** as part of lifestyle changes that also comprise cessation of smoking, increases in physical activity and reduction in body weight. In addition insulin, biguanides, acarbose and glitazones applied for glycemic control also improve diabetic dyslipidemia. Additional pharmacological treatment of dyslipidemia if persisting after glycemic control relies on different drug classes. Fibrates effectively reduce free fattyacids, fasting and postprandial lipemia, shift the distribution of LDL particles towards less dense subfractions and increase HDL cholesterol, thus particularly addressing key components of diabetic dyslipidemia. For LDL cholesterol lowering statins are mainly used that decrease LDL cholesterol levels by competitive inhibition of the HMG-CoA reductase. As **type 2 diabetes** is found to be associated with a two- to fourfold increase in coronary heart disease risk and as the degree of glycemia is more related to microvascular complications, correcting dyslipidemia appears to be a major task in order to reduce macrovascular events in patients with **type 2 diabetes**.

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ACCESSION NUMBER: 2002238815 EMBASE
TITLE: Type of dietary fat and insulin resistance.
AUTHOR: Rivellese A.A.; De Natale C.; Lilli S.
CORPORATE SOURCE: A.A. Rivellese, Department of Clinical Medicine, Federico II Univ. Medical School, Via S. Pansini 5, 80131 Napoli, Italy. rivelles@unina.it
SOURCE: Annals of the New York Academy of Sciences, (2002) Vol. 967, pp. 329-335.
Refs: 24
ISSN: 0077-8923 CODEN: ANYAA
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 003 Endocrinology
005 General Pathology and Pathological Anatomy
006 Internal Medicine
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20020725
Last Updated on STN: 20020725

ED Entered STN: 20020725
Last Updated on STN: 20020725

AB Animal studies have already shown the possibility to modulate insulin action by changing not only the amount of total fat, but also the type of fat. In these studies, saturated fat significantly increased insulin resistance, long- and short-chain **.omega.3 fatty acids** significantly improved it, whereas the effects of monounsaturated and ω6 polyunsaturated fatty acids ranged somewhere in between the two. A recent multicenter study (the Kanwu

study) on humans has shown that shifting from a diet rich in saturated fatty acids to one rich in monounsaturated fat improved insulin sensitivity in healthy people, while a moderate ω3 supplementation

lipid metabolism. With respect to blood pressure, the majority of studies show that **.omega.3 fatty acids** are able to reduce blood pressure in hypertensive patients, but not in normotensive individuals; this result has been confirmed also by the Kanwu study, where no changes in blood pressure were seen after ω3 supplementation in healthy people. On the other hand, in this study, the change from saturated to **monounsaturated fatty acids** was able to significantly reduce diastolic blood pressure. As to the lipid abnormalities more frequently present in the **metabolic** syndrome (i.e., hypertriglyceridemia and low HDL cholesterol), the main effects are related to **.omega.3 fatty acids**, which surely reduce **triglyceride** levels, but at the same time increase LDL cholesterol. In conclusion, there is so far sound evidence in humans that the quality of dietary fat is able to influence insulin resistance and some of the related **metabolic** abnormalities.

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ACCESSION NUMBER: 2001225097 EMBASE
TITLE: Postprandial **triglycerides** and endothelial function.
AUTHOR: Jagla A.; Schrezenmeir J.
CORPORATE SOURCE: Prof. J. Schrezenmeir, Inst. of Physiol./Biochem. of Nutr., Federal Research Centre, Hermann-Weigmann-Str. 1, D-24116 Kiel, Germany. schrezenmeir@bafm.de
SOURCE: Experimental and Clinical Endocrinology and Diabetes, (2001) Vol. 109, No. 4, pp. S533-S547.
Refs: 149
ISSN: 0947-7349 CODEN: ECEDFQ
COUNTRY: Germany
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 003 Endocrinology
018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20010717
Last Updated on STN: 20010717

ED Entered STN: 20010717
Last Updated on STN: 20010717

AB Several studies support the association between postprandially elevated **triglyceride** levels and atherosclerosis. Histological and cell culture investigations revealed, that **triglyceride** rich postprandial lipoproteins are taken up by macrophages and smooth muscle cells and are detectable as part of foam cells in vascular lesions. Remnant particles, generated by lipolysis of postprandial lipoproteins in vitro and fatty acids increase the permeability of the endothelium and are cytotoxic for endothelial cells. - Besides these morphological changes of cells, lipoproteins have been shown to exert effects on cellular functions like the expression of membrane proteins and the production or release of several bioactive substances regulating communication with blood cells and other cell systems of the vascular wall, blood pressure and hemostasis. - This review concentrates on the influence of postprandial lipoproteins on factors involved in the interaction of endothelial cells with blood leukocytes and factors mediating blood pressure regulation. Increased expression of adhesion molecules has been detected immunehistochemically

in atherosclerotic plaques in animals and humans. It was demonstrated that patients with elevated **triglyceride** levels have increased levels of soluble adhesion molecules. Furthermore, postprandial **linoleic acid**, induced higher adhesion molecule expression at higher oxidant concentration compared with chylomicrons separated after ingestion of **olive oil**, rich in monounsaturated **oleic acid**. - Several authors described effects of fatty acids on the expression of adhesion molecules. On the one hand, they may exert stimulatory effects as such, on the other hand cytokine induced adhesion molecule expression may be enhanced by certain fatty acids and inhibited by others, implying an interference with signal transduction processes. - Effects of lipoproteins on vasoactive substances seem to be implicated in endothelial dysfunction, too. The endothelium-derived relaxing factor nitric oxide (NO) has gained increasingly attention in the last two decades and is regarded as protective against hypertension and atherosclerosis. It was demonstrated that chylomicrons and their remnants inhibited endothelium-dependent relaxations in isolated aortas. Vasodilatatory responses and nitric oxide **metabolism** were shown to be affected by the amount and composition of dietary fat. Cell culture experiments revealed modulation of NO release by certain fatty acids. - Plasma levels of endothelin-1, a strong vasoconstrictor, have been shown to be increased in patients with **type 2 diabetes** and **metabolic** syndrome, respectively. Postprandially elevated **triglycerides** increased endothelin-levels in addition to insulin in patients with **metabolic** syndrome. - In summary, there is evidence that the association between postprandial **triglycerides** and the **metabolic** syndrome is driven by direct influences on endothelial functions because plasma **triglyceride** levels are associated with levels of humoral risk markers of endothelial origin, and postprandial lipoproteins stimulate the release and/or expression of endothelial mediators in vitro, which induce atherogenesis and hypertension.

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ACCESSION NUMBER: 2000337269 EMBASE

TITLE: Long-chain acyl-CoA esters as indicators of **lipid metabolism** and insulin sensitivity in rat and human muscle.

AUTHOR: Ellis B.A.; Poynten A.; Lowy A.J.; Furler S.M.; Chisholm D.J.; Kraegen E.W.; Cooney G.J.

CORPORATE SOURCE: B.A. Ellis, Garvan Inst. of Medical Research, 384 Victoria St., Darlinghurst, NSW 2010, Australia. b.ellis@garvan.unsw.edu.au

SOURCE: American Journal of Physiology - Endocrinology and Metabolism, (2000) Vol. 279, No. 3 42-3, pp. E554-E560. Refs: 36
ISSN: 0193-1849 CODEN: AJPMD

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 002 Physiology
003 Endocrinology
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 20001013
Last Updated on STN: 20001013

ED Entered STN: 20001013
Last Updated on STN: 20001013

AB Long-chain acyl-CoAs (LCACoA) are an activated lipid species that are key

metabolites in **lipid metabolism**; they also have a role in the regulation of other cellular processes. However, few studies have linked LCACoA content in rat and human muscle to changes in

0.0001) by specifically increasing 18:2-CoA. The LCACoA content of red muscle from rats (4-8 nmol/g) was 4- to 10-fold higher than adipose tissue (0.4-0.9 nmol/g, P < 0.001), suggesting that any contamination of muscle samples with adipocytes would contribute little to the LCACoA content of muscle. In humans, the LCACoA content of muscle correlated significantly with a measure of whole body insulin action in 17 male subjects (r2 = 0.34, P = 0.01), supporting a link between muscle **lipid metabolism** and insulin action. These results demonstrate that the LCACoA pool reflects **lipid metabolism** and nutritional state in muscle. We conclude that the LCACoA content of muscle provides a direct index of intracellular **lipid metabolism** and its links to insulin action, which, unlike **triglyceride** content, is not subject to contamination by closely associated adipose tissue.

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ACCESSION NUMBER: 97346372 EMBASE
DOCUMENT NUMBER: 1997346372
TITLE: Monounsaturated and marine .**omega**.-**3** **fatty acids** in NIDDM patients.
AUTHOR: Rivellese A.A.
CORPORATE SOURCE: A.A. Rivellese, Dept. Clinical Experimental Medicine, Federico II University Medical Sch., 80131 Naples, Italy
SOURCE: Annals of the New York Academy of Sciences, (1997) Vol. 827, pp. 302-309.
Refs: 24
ISSN: 0077-8923 CODEN: ANYAA
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 003 Endocrinology
017 Public Health, Social Medicine and Epidemiology
029 Clinical Biochemistry
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered STN: 971201
Last Updated on STN: 971201
ED Entered STN: 971201
Last Updated on STN: 971201
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L37 ANSWER 49 OF 56 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 97262390 EMBASE
DOCUMENT NUMBER: 1997262390
TITLE: Balanced intakes of natural **triglycerides** for optimum nutrition: An evolutionary and phytochemical perspective.
AUTHOR: Broadhurst C.L.
CORPORATE SOURCE: C.L. Broadhurst, 22nd Century Nutrition, Inc., Herbal Vineyard, Inc., 1315 Harding Ln, Cloverly, MD 20905-4007, United States. cleigh@cais.com
SOURCE: Medical Hypotheses, (1997) Vol. 49, No. 3, pp. 247-261.
Refs: 117
ISSN: 0306-9877 CODEN: MEHYDY
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry

LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 970212

balance of polyunsaturated fat, monounsaturated fat, and saturated fat. Since we are still a Paleolithic species, adapted to eating only wild foods, it is difficult to justify the consumption of anything other than an overall balance of **triglyceride**/phospholipid types in an evolutionary sense. No natural fats are intrinsically good or bad - it is the proportions that matter. Variety is recommended in dietary lipid structure, degree of saturation, and chain length. Pathological n-3/n-6 polyunsaturated fat imbalance, obesity, and progressive **glucose intolerance** are consequences of adopting cereal grain based diets by both humans and livestock. Food processing and refining amplify these problems. Excessive concerns regarding polyunsaturated fat peroxidation in vivo are not warranted when trigylcerides are balanced and normal diets are consumed. Numerous phytochemicals present in unrefined oils, fruits, vegetables, and herbs afford significant protection from lipid peroxidation and chronic disease.

L37 ANSWER 50 OF 56 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 96253804 EMBASE
DOCUMENT NUMBER: 1996253804
TITLE: Effects of inhibiting cholesterol absorption and synthesis on cholesterol and lipoprotein **metabolism** in hypercholesterolemic non-insulin- dependent diabetic men.
AUTHOR: Gylling H.; Miettinen T.A.
CORPORATE SOURCE: Division of Internal Medicine, Department of Medicine, University of Helsinki, Haartmaninkatu 4,FIN-00290 Helsinki, Finland
SOURCE: Journal of Lipid Research, (1996) Vol. 37, No. 8, pp. 1776-1785.
ISSN: 0022-2275 CODEN: JLPRAW
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 961001
Last Updated on STN: 961001
ED Entered STN: 961001
Last Updated on STN: 961001
AB Effectiveness of a simultaneous inhibition of cholesterol absorption and synthesis, caused by sitostanol ester margarine and pravastatin, was studied to control mild hypercholesterolemia in men with non-insulin-dependent **diabetes mellitus** (NIDDM) (n = 8). Margarine, 24 g daily, was a basal dietary treatment. Four 7-week intervention periods included margarine, sitostanol (3 g/day) ester margarine, pravastatin (40 mg/day), and sitostanol ester margarine plus pravastatin in a random order. Pravastatin lowered serum total (-32%) and LDL cholesterol (-38%) and apolipoprotein B (-39%) because of enhanced removal (+20%) and decreased production (-26%) of LDL apolipoprotein B, and reduced synthesis (-9%) and turnover (-8%) of cholesterol, which resulted in reduced biliary cholesterol secretion (-18%). Even though serum **triglycerides** were lowered by 28%, VLDL, IDL, and light and dense LDL became **triglyceride**-enriched. Despite increasing cholesterol synthesis, sitostanol lowered LDL cholesterol (-14%) by

inhibiting cholesterol absorption (-68%) and LDL apolipoprotein B production rate (-20%). Combination of pravastatin and sitostanol ester lowered serum total, VLDL, IDL, and LDL cholesterol and LDL apolipoprotein

unchanged. In spite of decreased absorption, cholesterol synthesis was not compensatorily increased. In conclusion, simultaneous inhibition of cholesterol absorption and synthesis lowers LDL cholesterol and apolipoprotein B by 44-45% solely through inhibition of LDL apolipoprotein B production rate in hypercholesterolemic NIDDM patients. A combination of statin to sitostanol ester margarine-resistant patients offers a safe and effective measure to normalize abnormally high cholesterol values, probably with a lowered statin dose.

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ACCESSION NUMBER: 94380564 EMBASE
DOCUMENT NUMBER: 1994380564
TITLE: Dietary substitution of **medium chain triglycerides** in subjects with non-insulin-dependent **diabetes mellitus** in an ambulatory setting: Impact on glycemic control and insulin-mediated glucose **metabolism**.
AUTHOR: Yost T.J.; Erskine J.M.; Gregg T.S.; Podlecki D.L.; Brass E.P.; Eckel R.H.
CORPORATE SOURCE: Colorado Univ. Health Sciences Ctr., 4200 East Ninth Avenue B151,Denver, CO 80262, United States
SOURCE: Journal of the American College of Nutrition, (1994) Vol. 13, No. 6, pp. 615-622.
ISSN: 0731-5724 CODEN: JONUDL
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 950112
Last Updated on STN: 950112
ED Entered STN: 950112
Last Updated on STN: 950112

AB Objective: We have previously shown in an acute inpatient setting that dietary substitution of 77.5% of fat kcal as **medium chain triglycerides** (**MCT**) increased insulin-mediated glucose **metabolism** in patients with non-insulin- dependent **diabetes mellitus** (NIDDM), and that this effect appeared to be mediated by increases in insulin-mediated glucose disposal. The purpose of this study was to test the application of dietary substitution of **medium chain triglycerides** as an adjunctive tool in ambulatory therapy of NIDDM. Methods: Five subjects with NIDDM underwent a baseline 6 hour insulin/glucose euglycemic clamp study, with simultaneous 3H-glucose infusion for calculation of glucose disposal rate and hepatic glucose output. Subjects were then randomized to begin one of two 30-day experimental diets, with long chain (LCT) or **medium chain triglycerides** (**MCT**), and subsequent crossover to the other diet. A 6 hour euglycemic clamp was repeated after each diet phase. Results: Diet records and urinary organic acid excretion indicated a high level of dietary compliance by the study participants. Postprandial blood glucose excursions were less after one month on the diet with **MCT** than after the LCT diet ($p = 0.004$). However,

fasting serum glucose, serum fructosamine (a measure of glycemia), fasting insulin, hepatic glucose output, and insulin-mediated glucose

L37 ANSWER 52 OF 56 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 93084217 EMBASE
DOCUMENT NUMBER: 1993084217
TITLE: Dietary lipid profile is a determinant of tissue phospholipid fatty acid composition and rate of weight gain in rats.
AUTHOR: Pan D.A.; Storlien L.H.
CORPORATE SOURCE: Department Medicine [Endocrinology], University of Sydney,Sydney, NSW 2006, Australia
SOURCE: Journal of Nutrition, (1993) Vol. 123, No. 3, pp. 512-519. ISSN: 0022-3166 CODEN: JONUAI
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 930425
Last Updated on STN: 930425
ED Entered STN: 930425
Last Updated on STN: 930425
AB Modifications in membrane fatty acid composition and insulin action are possible through dietary intervention. We examined the **metabolic** fate of (n- 3) fatty acids in male Wistar rats, using three isocaloric, high fat diets. The ET-L, OL-L and SAF-L diets contained edible tallow, **olive oil** and safflower oil, respectively, with identical amounts of (n-3) fatty acids as **linseed oil**. Despite isocaloric feeding, weight gain was lower ($P < 0.001$) in rats fed the more highly saturated ET-L diet (69 ± 8 g) than in those fed either the high (n-9) fatty acid OL-L diet (93 ± 2 g) or the high (n-6) fatty acid SAF-L diet (108 ± 4 g). Analysis of red quadricep fatty acid composition revealed phospholipid (n-3) fatty acid levels in the ET-L-fed group (21.6 ± 0.8 g/100 g fatty acids) to be significantly higher than in either the OL-L-fed (17.7 ± 0.6 g/100 g fatty acids, $P < 0.05$) or SAF-L-fed (15.3 ± 0.7 g/100 g fatty acids, $P < 0.05$) group. A similar pattern was observed in other muscles and white adipose tissue. A follow-up study using 14C-labeled (n-3) fatty acids in the diet showed greater (n-3) fatty acid incorporation in the ET-L-fed group relative to the other two groups and conversely lower 14CO2 production than in the SAF-L-fed group. These results demonstrate that **metabolic** fate of dietary fatty acids is strongly influenced by the overall fatty acid profile of the diet. The functional consequences are seen in the differing rates of weight gain despite equal intakes, with tissue (n-3) fatty acid apparently protective against weight gain. Because obesity is a powerful predictor of insulin resistance, these results have implications for dietary treatment of diabetes.

L37 ANSWER 53 OF 56 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 77037367 EMBASE
DOCUMENT NUMBER: 1977037367
TITLE: Time course of changes in blood glucose and ketone bodies, plasma lipids and liver fatty acid composition in streptozotocin diabetic male rats.
AUTHOR: Topping D.L.; Targ M.E.

CORPORATE SOURCE: Dept. Biochem. Clin. Chem., Hazleton Lab. Europe Ltd, Harrogate, United Kingdom

SOURCE: Hormone Research (1975) Vol. 6, No. 3, pp. 122-137

LANGUAGE: English

AB Male rats were given streptozotocin (100 mg/kg) by intraperitoneal injection. Groups of control and streptozotocin treated animals were killed at daily intervals for 4 days after injection. Over this period, treated rats lost weight continuously while control animals progressively gained weight. Within 24 h of treatment blood glucose and plasma free acids were raised to levels which were sustained for the remainder of the experiment. After 48 h blood ketone bodies, plasma cholesterol and **triglycerides** were maximally raised and liver glycogen and blood lactate similarly lowered. The percentage composition of major fatty acids in liver lipids was unchanged until 4 days after treatment when there were significant increases in the proportion of **oleate** and **linoleate** and reductions in stearate and arachidonate. The data confirm that streptozotocin induces a rapid and sustained diabetes. It is suggested that **metabolic** experiments, in streptozotocin diabetic rats, may be performed 48 h after treatment.

L37 ANSWER 54 OF 56 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 75053725 EMBASE

DOCUMENT NUMBER: 1975053725

TITLE: [**Medium chain triglycerides**. Clinical physiology and applications]. MITTELKETTIGE **TRIGLYCERIDE**. KLINISCHE PHYSIOLOGIE UND ANWENDUNG.

AUTHOR: Sailer D.; Berg G.

CORPORATE SOURCE: Forsch. Abt. Ernahr. Stoffwechselkrankh., Med. Klin., Poliklin., Univ. Erlangen/Nurnberg, Germany

SOURCE: Zeitschrift fur Ernahrungswissenschaft, (1974) Vol. 13, No. 1-2, pp. 6-17.
CODEN: ZERNAL

DOCUMENT TYPE: Journal

FILE SEGMENT: 037 Drug Literature Index
029 Clinical Biochemistry
006 Internal Medicine
017 Public Health, Social Medicine and Epidemiology

LANGUAGE: German

AB **Medium chain triglycerides** differ in their biochemical and physical properties from the usual food fats. The considerable independence of pancreatic lipase and bile acids, rapid absorption and the rapid provision of energy make the chain **triglycerides** a valuable dietetic in modern nutrition in all diseases associated with malabsorption of fats and lymph drainage disturbances. They can also be successfully used in fat induced hyperlipidemia and hypercholesterolemia. The clinical value of these fats and also the most varied **metabolic** effects are discussed.

L37 ANSWER 55 OF 56 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

ACCESSION NUMBER: 2004-422255 [40] WPIDS

DOC. NO. CPI: C2004-158703

TITLE: Dietetic therapy of **diabetes mellitus** in adults or adolescents, using **medium-chain triglycerides**, e.g. incorporated in margarine or edible oil, to regulate and optimize **metabolism**.

DERWENT CLASS: B05 D13

INVENTOR(S): HEIRLER, H

PATENT ASSIGNEE(S): (HEIR-N) HEIRLER PROJEKTE ERNAEHRUNG MEDIZIN OEKO
COUNTRY COUNT: 32
PATENT INFORMATION:

R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV
MC MK NL PT RO SE SI SK TR
DE 10254584 A1 20040609 (200440)
US 2004151757 A1 20040805 (200452)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 1421858	A1	EP 2003-26659	20031119
DE 10254584	A1	DE 2002-10254584	20021122
US 2004151757	A1	US 2003-717990	20031121

PRIORITY APPLN. INFO: DE 2002-10254584 20021122
ED 20040624
AN 2004-422255 [40] WPIDS
AB EP 1421858 A UPAB: 20040624
NOVELTY - The use of **medium-chain triglycerides** (I), or a (I)-containing composition (A), for the treatment of **diabetes mellitus**.
ACTIVITY - Antidiabetic; Anabolic.
MECHANISM OF ACTION - None given.
USE - For treatment of adults or adolescents with **diabetes mellitus** by supplemented, balanced nutrition and diet. The (I)-containing compositions are typically in the form of margarine or edible oil (e.g. for use in salads, soups, dressings or ketchups or as frying or cooking oil).
ADVANTAGE - (I) regulate and optimize the **metabolic** situation in diabetes patients. (I) proceed in association with the lymph pathway via the portal vein directly into the liver to be oxidized; are not accumulated in fatty tissue; and have a lower calorific value (8.3 kcal/g) than fats with long-chain fatty acids (9.3 kal/g).
Dwg.0/0

L37 ANSWER 56 OF 56 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
ACCESSION NUMBER: 2002-083157 [11] WPIDS
DOC. NO. CPI: C2002-025270
TITLE: Oil composition for treating cardiovascular diseases e.g. atherosclerosis and hyperlipidemia, comprises **triglycerides** bearing short, medium and long chain fatty acid residues optionally with phytosterol.
DERWENT CLASS: B04 D13 D23
INVENTOR(S): JONES, P J; ZAWISTOWSKI, J
PATENT ASSIGNEE(S): (FORB-N) FORBES MEDI-TECH INC
COUNTRY COUNT: 88
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001091587	A2	20011206	(200211)*	EN	84

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZA ZW

AU 2001065722 A 20011211 (200225)
JP 2003534356 W 20031118 (200401) 63
EP 1408775 A2 20040421 (200427) EN

PATENT NO	KIND	APPLICATION	DATE
WO 2001091587	A2	WO 2001-CA802	20010604
AU 2001065722	A	AU 2001-65722	20010604
JP 2003534356	W	JP 2001-587608	20010604
		WO 2001-CA802	20010604
EP 1408775	A2	EP 2001-942929	20010604
		WO 2001-CA802	20010604

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001065722	A	Based on	WO 2001091587
JP 2003534356	W	Based on	WO 2001091587
EP 1408775	A2	Based on	WO 2001091587

PRIORITY APPLN. INFO: US 2000-586431 20000602
ED 20020215
AN 2002-083157 [11] WPIDS
AB WO 200191587 A UPAB: 20020215
NOVELTY - An oil composition comprises **triglycerides** bearing short and medium chain fatty acid residues derived from fatty acids having 4-14 C and long chain fatty acid residues derived from fatty acids having 15-22 C.

ACTIVITY - Antiarteriosclerotic; antilipemic; hypotensive; anticoagulant; thrombolytic; antidiabetic; nootropic; cytostatic.

MECHANISM OF ACTION - None given.

USE - For treating and preventing cardiovascular diseases and its underlying conditions including atherosclerosis, hypercholesterolemia (claimed), hyperlipidemia, hypertension, thrombosis and related diseases such as **Type II diabetes** as well as other diseases that include oxidative damage such as dementia, ageing and cancer in mammal (preferably human).

ADVANTAGE - The composition reduces weight gain and maintains proper body weight via the enhanced **metabolism** of fats and decreased energy expenditure, lowers serum cholesterol and **triglycerides** Dwg.0/35

=> d cost

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
CONNECT CHARGES	15.33	155.66
NETWORK CHARGES	0.60	7.62
DISPLAY CHARGES	102.25	226.40
FULL ESTIMATED COST	118.18	389.68

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-10.95	-26.28

IN FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS'
AT 11:59:11 ON 22 APR 2005

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=> d his

     (FILE 'HOME' ENTERED AT 10:45:15 ON 22 APR 2005)

     FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS' ENTERED AT 10:46:14 ON 22
     APR 2005
L2        624438 S (DIABETES(W)MELLITUS) OR "TYPE 1 DIABETES" OR "TYPE 2 DIABETE
L3        316701 S (MEDIUM(W)CHAIN(W)TRIGLYCERIDE?) OR TRIACYLGLYCEROL? OR TRIGL
L4        198146 S (OLEIC ACID?) OR "9-OCTADECENOIC ACID" OR OLEATE? OR (OLIVE O
L5        122681 S (LINOLEIC ACID?) OR LINOLEATE? OR "9,12-OCTADECADIENOIC ACID"
L6          2949 S (Ω)(W)"6"(W)FATTY(W)ACID? OR (DOUBLE(W)UNSATURATED(W)TR
L7         85651 S ((A)(W)LINOLENIC(W)ACID?) OR (LINOLENIC ACID?) OR LINOL
L8             1 S (TRIPLE(W)UNSATURATED(W)TRIGLYCERIDE?)
L9         52757 S EICOSAPENTAEN? OR EICOSAPENTAENOIC ACID? OR TIMNODONIC ACID?
L10        60428 S DOCOSAHEXAEN? OR DOCOSAHEXAENOIC ACID? OR "DHA" OR (FISH OIL?
L11         4405 S (SATURATED(W)LONG(W)CHAIN(W)(TRIGLYCERIDE? OR TRIACYLGLYCEROL
L12       152425 S EMULSIFIER OR EMULSIF?
L13        69467 S MONOGLYCERIDE? OR MONOACYLGLYCEROL? OR DIGLYCERIDE? OR DIACYL
L14       421736 S "VITAMIN A" OR RETINOL? OR RETINYL PALMITATE? OR "VITAMIN D"
L15        48454 S CAROTIN? OR (BETA(W)(CAROTIN? OR CAROTENE?)) OR ((B)(W)
L16        27388 S FLAVORING? OR (BUTTER(W)FLAVOR?) OR ROSEMARY? OR (ROSEMARY(W)
L17       130972 S RETINYL PALMITATE? OR "VITAMIN D3" OR CHOLECALCIFEROL? OR "VI
L18       197160 S "VITAMIN B6" OR PICOLINE? OR PYRIDOXINE? OR PYRIDOXAL? OR PYR
L19       404698 S "VITAMIN C" OR ASCORBIC ACID? OR ASCORBYL PALMITATE? OR "VITA
L20      4846639 S ZINC? OR CHROM? OR MANGANESE?
L21        32231 S L2 AND L3
L22          699 S L21 AND L4
L23          204 S L22 AND (L5 OR L6 OR L7 OR L8)
L24           73 S L23 AND (L9 OR L10)
L25            1 S L24 AND L11
L26           14 S L24 AND (L12 OR L13 OR L14 OR L15 OR L16)
L27           11 DUP REM L26 (3 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 11:18:33 ON 22 APR 2005
L28            0 S L24 NOT L26

     FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS' ENTERED AT 11:22:48 ON 22
     APR 2005
L29           59 S L24 NOT L26
L30           40 DUP REM L29 (19 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 11:23:32 ON 22 APR 2005

     FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS' ENTERED AT 11:53:31 ON 22
     APR 2005
L31      7724971 S METABOL? OR (FAT METABOL?) OR (LIPID METABOL?) OR (FATTY ACID
L32          453 S L22 AND L31
L33          101 S L23 AND L31
L34           80 DUP REM L33 (21 DUPLICATES REMOVED)
L35           62 S L34 NOT L30
L36           56 S L35 NOT L27
L37           56 DUP REM L36 (0 DUPLICATES REMOVED)
L38          453 S L22 (L) L31

=> save
ENTER L#, L# RANGE, ALL, OR (END):all
ENTER NAME OR (END):110717990/l
L# LIST L1-L38 HAS BEEN SAVED AS 'L10717990/L'

=> d his
```

(FILE 'HOME' ENTERED AT 10:45:15 ON 22 APR 2005)

FILE 'MEDLINE, BIOSIS, HCAPLUS, EMBASE, WPIDS' ENTERED AT 10:46:14 ON 22 APR 2005

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L2        624438 S (DIABETES(W)MELLITUS) OR "TYPE 1 DIABETES" OR "TYPE 2 DIABETE
L3        316701 S (MEDIUM(W)CHAIN(W)TRIGLYCERIDE?) OR TRIACYLGLYCEROL? OR TRIGL
L4        198146 S (OLEIC ACID?) OR "9-OCTADECENOIC ACID" OR OLEATE? OR (OLIVE O
L5        122681 S (LINOLEIC ACID?) OR LINOLEATE? OR "9,12-OCTADECADIENOIC ACID"
L6          2949 S (Ω)(W)"6"(W)FATTY(W)ACID? OR (DOUBLE(W)UNSATURATED(W)TR
L7         85651 S ((A)(W)LINOLENIC(W)ACID?) OR (LINOLENIC ACID?) OR LINOL
L8             1 S (TRIPLE(W)UNSATURATED(W)TRIGLYCERIDE?)
L9         52757 S EICOSAPENTAEN? OR EICOSAPENTAENOIC ACID? OR TIMNODONIC ACID?
L10        60428 S DOCOSAHEXAEN? OR DOCOSAHEXAENOIC ACID? OR "DHA" OR (FISH OIL?
L11         4405 S (SATURATED(W)LONG(W)CHAIN(W)(TRIGLYCERIDE? OR TRIACYLGLYCEROL
L12       152425 S EMULSIFIER OR EMULSIF?
L13        69467 S MONOGLYCERIDE? OR MONOACYLGLYCEROL? OR DIGLYCERIDE? OR DIACYL
L14       421736 S "VITAMIN A" OR RETINOL? OR RETINYL PALMITATE? OR "VITAMIN D"
L15        48454 S CAROTIN? OR (BETA(W)(CAROTIN? OR CAROTENE?)) OR ((B)(W)
L16        27388 S FLAVORING? OR (BUTTER(W)FLAVOR?) OR ROSEMARY? OR (ROSEMARY(W)
L17       130972 S RETINYL PALMITATE? OR "VITAMIN D3" OR CHOLECALCIFEROL? OR "VI
L18       197160 S "VITAMIN B6" OR PICOLINE? OR PYRIDOXINE? OR PYRIDOXAL? OR PYR
L19       404698 S "VITAMIN C" OR ASCORBIC ACID? OR ASCORBYL PALMITATE? OR "VITA
L20      4846639 S ZINC? OR CHROM? OR MANGANESE?
L21        32231 S L2 AND L3
L22          699 S L21 AND L4
L23          204 S L22 AND (L5 OR L6 OR L7 OR L8)
L24           73 S L23 AND (L9 OR L10)
L25            1 S L24 AND L11
L26           14 S L24 AND (L12 OR L13 OR L14 OR L15 OR L16)
L27           11 DUP REM L26 (3 DUPLICATES REMOVED)
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L28            0 S L24 NOT L26
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L32          453 S L22 AND L31
L33          101 S L23 AND L31
L34           80 DUP REM L33 (21 DUPLICATES REMOVED)
L35           62 S L34 NOT L30
L36           56 S L35 NOT L27
L37           56 DUP REM L36 (0 DUPLICATES REMOVED)
L38          453 S L22 (L) L31
                 SAVE ALL L10717990/L
```